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PROCEEDINGS

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Section for the Study of Disease in Children.

President—Dr. J. D. ROLLESTON.

Two Cases of (?) Still's Disease.

By W. S. C. COPEMAN, M.B.

Case I.—I. G., AGED 9 years, came to Hospital for Sick Children in March, 1925, complaining of pains in neck, knees and wrists for fourteen days. The wrists were considerably swollen, and an urticarial rash was marked over both elbows, and on the inner side of the knees, especially the left. The case was diagnosed as rheumatoid arthritis. The tonsils appeared to be normal, and the spleen was not enlarged. The heart showed no abnormality, but the epitrochlear glands were palpable.

Patient was re-admitted with same symptoms, August-October, 1926, when, in addition, the elbows were found to be swollen, and the cervical and axillary glands to be enlarged. A Flexner bacillus was found in the stools.

Patient was seen on March 3, 1927, in the Out-patient Department, when the report was that the wrists were still swollen, with some fluid over the dorsum. Other joints normal. Epitrochlear glands not palpable, and axillary and cervical glands much smaller. Patient was re-admitted on next day for further investigation.

Condition on Admission.—Well-nourished child, good colour. Wrists swollen, not red, hot or painful. No swelling of knees or elbows. Extension and flexion of wrists much limited by (?) peri-articular swelling. Heart normal. Small axillary, cervical and inguinal glands.

Respiratory system normal. Tongue clean; teeth good.

Abdomen.—Liver, spleen and kidney not palpable. No tenderness or rigidity.

Nervous system normal.

Urine.—Acid, 1020. Nothing abnormal. Sterile.

Ten days after admission wrists became more swollen and painful: pain only lasted for one day.

Flexner bacillus not now found in stool. Fat-content ratio of stool normal.

Lævulose blood-sugar test: before lævulose 0·100 per cent.; one hour after, 0·123 per cent.; two hours after, 0·126 per cent.

X-ray.—"Some rarefaction in carpal bones with some defined areas also in bases of metacarpals—so-called 'drill-holes.' Flattening of radial epiphyses with some sclerosis of articular surface. Appearances typical of Still's disease."

Case II.—E. B., aged 6½ years. Admitted under Mr. Fairbank to Surgical Ward of Hospital for Sick Children in July, 1925, with painless swelling of the knees and genu valgum of three years' duration. This had been treated in plaster for fifteen months

? Still's Disease (Case II).
FIG. 1.

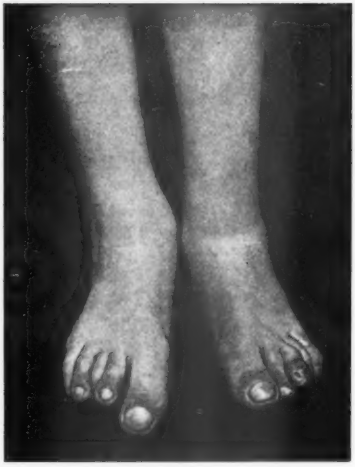


FIG 2.

with no improvement: diagnosed as tuberculous. Other joints beginning to enlarge slightly.

Operation: Left knee, June 23, 1925. Only very small quantity of clear fluid found. Piece of synovial membrane excised.

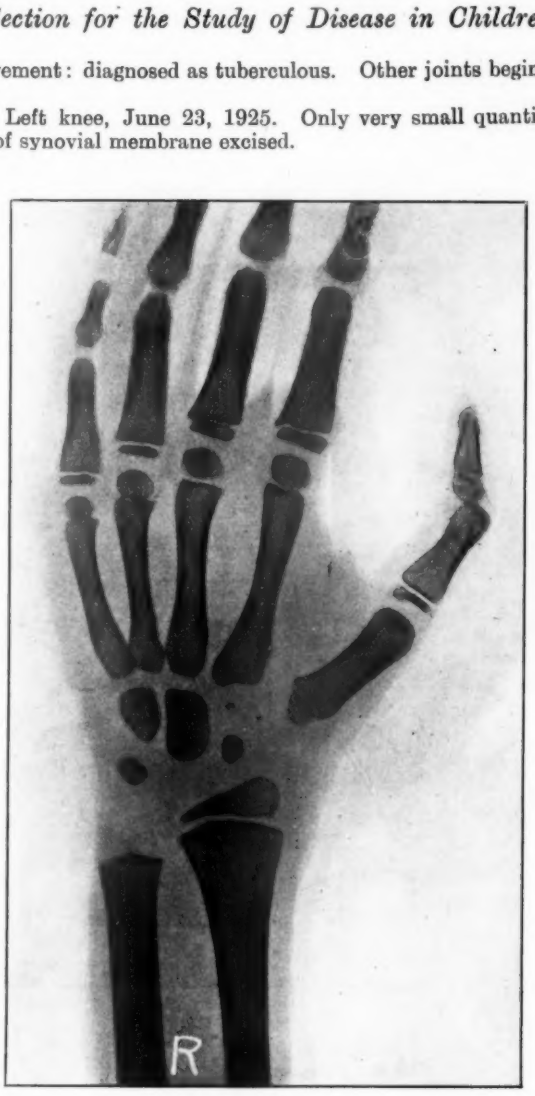


FIG. 3.

Section showed no evidence of pathological change. Fluid was inoculated into guinea-pigs, and no sign of tuberculosis found, post-mortem, six weeks later.

Re-admitted March 7, 1927, under Dr. Poynton: mother stated that during the previous six weeks the joints had become more swollen. There is no history of any febrile condition preceding this. There is no pain, and patient has been attending school.

40 Copeman: *Two Cases of (?) Still's Disease*

Condition on Admission.—Thin, cheerful, good coloured child, "always has been delicate," winter cough. Has had measles, whooping-cough and bronchitis. Eldest of three children. Mother is said to be rheumatic. Family live in two rooms which may be damp.

There is marked "spatula" clubbing of the fingers of both hands, enlargement of knees, ankles and wrists, and slight enlargement of the elbows. There is no tenderness,

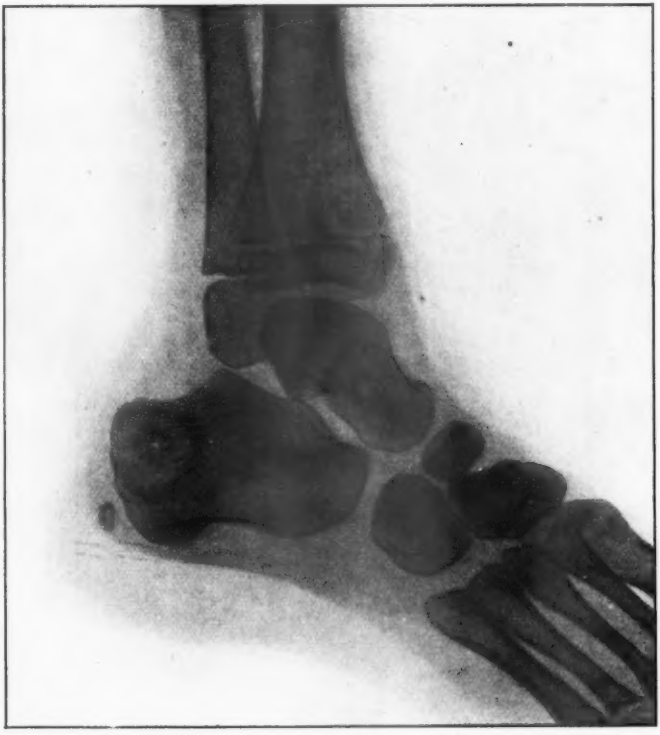


FIG. 4.

limitation of movement, or crepitus. There is a tendency to pes valgus; "spatula" clubbing of toes. Enlarged glands in both axillæ. No injection of fauces.

Heart normal. *Respiratory System.*—Nothing abnormal detected. *Abdomen* normal, spleen not palpable. *Nervous system* normal. *Urine* slightly turbid, alkaline; amorphous phosphates in large quantities. Nothing otherwise abnormal. Sterile.

Seen by Dr. Poynton March 9, 1927. Swelling described as being probably due to a hypertrophic condition of synovial membrane. Patient noticed to be somewhat cyanosed.

X-ray.—"Much joint swelling. No marked bone changes. Appearances not

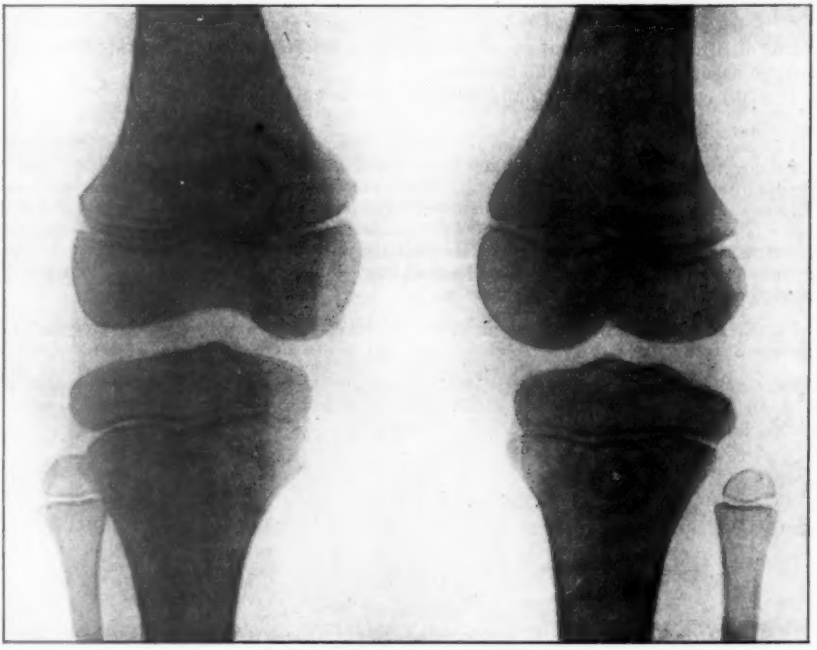


FIG. 5.

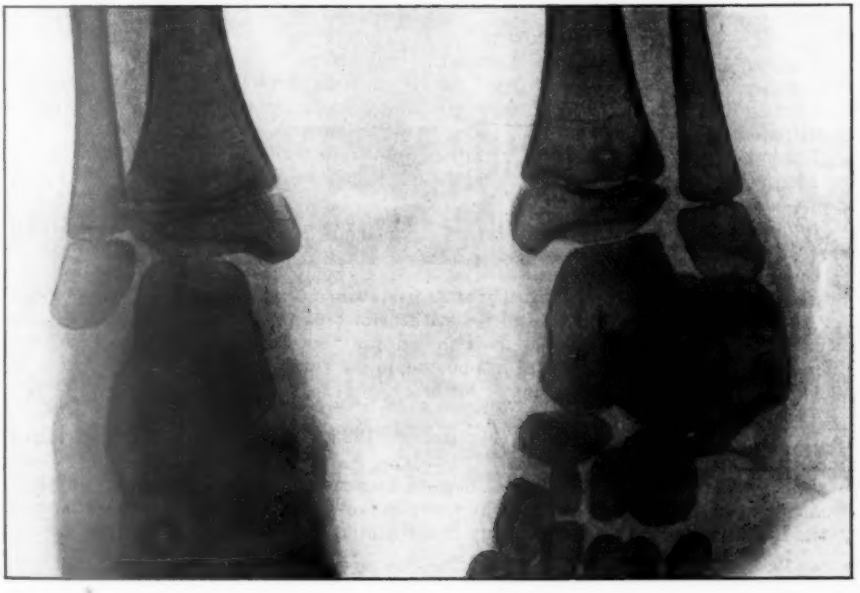


FIG. 6.

typical of Still's disease—but epiphysis of radius and epiphyses at ankle-joints are of the type associated with Still's disease."

In both cases the Wassermann reaction was negative.

Discussion.—Dr. NEILL HOBHOUSE said he was much interested in the note on the bacteriology, as he had wanted for some time to find out whether there was a relation between arthritis in children and bowel organisms. These resembled cases of dysenteric arthritis such as he saw in the Mediterranean regions during the war. The fluid in the joints in those cases was always sterile, but agglutinated Shiga's bacillus. He thought that light might be thrown on the possible relationship of arthritis to the microbial inhabitants of the bowel if those concerned with such cases would take the fluid from the affected joints and ascertain whether it agglutinated Shiga's or Flexner's bacillus.

Dr. E. A. COCKAYNE said he thought the second of these two cases was entirely different from one of Still's disease. The condition must be due to a congenital defect of some kind.

Dr. COPEMAN (in reply) said that Dr. Nabarro was investigating these cases on the lines which Dr. Hobhouse had just suggested. The blood had at one time agglutinated Flexner's bacillus, but did not do so now.

CLINICAL MEETING HELD AT THE VICTORIA HOSPITAL FOR CHILDREN, APRIL 29, 1927.

CASES.

Two Cases of Spasmodic Stricture of Œsophagus.

By R. C. JEWESBURY, M.D.

Case I.—Girl, aged 11 years.

History.—Infancy—delicate, often had attacks of vomiting.

October, 1920.—Aged 4½ years. Admitted with persistent vomiting. Could take fluids, but solids were returned immediately after swallowing. Very thirsty. Constipated. Temperature, pulse, and respiration normal.

December.—Œsophagoscopy: stricture 8 in. from teeth. Trachea normal.

Progress.—Gradual dilatation, after which solids were retained and child began to put on weight.

Subsequent dilatations (one sitting).—1921, two; 1922, two; 1923, one; 1924, one; 1925, two; 1926, one; 1927, March.

Case II.—Girl, aged 5 years.

History.—Often vomiting directly after feeds since birth.

August, 1922.—Aged 2½. Admitted with persistent vomiting. Solids and liquids returned immediately. Doubled up with pain in the epigastrium before vomiting. Bismuth meal showed stricture of œsophagus in its lower third.

October 1922.—Œsophagoscopy: stricture.

Progress.—Gradual dilatation.

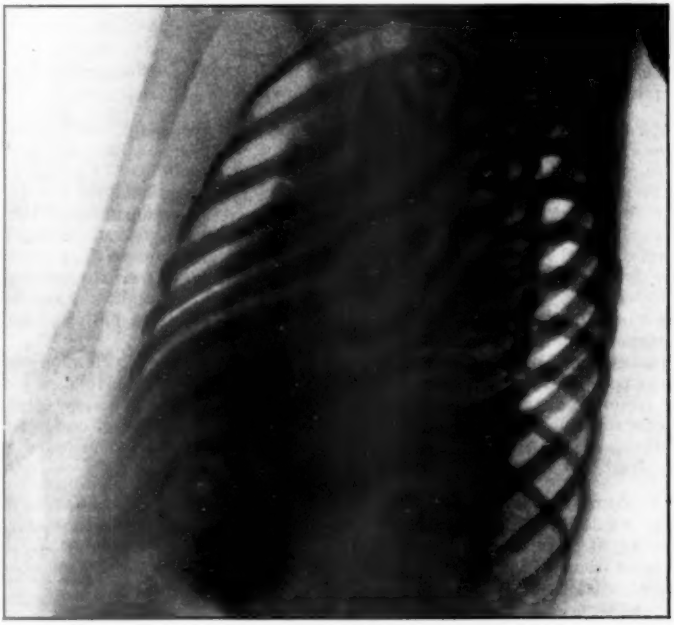
Subsequent dilatations.—1922, November; 1923, March, June. None required since.

I have brought these two cases forward because, as far as I can make out, this condition is extraordinarily rare in children; in fact I have not been able, in referring to the literature, to find records of any other similar cases of spasm, or whatever it

is preferred to call it, in children. It seems to be comparatively rare even in adults.

The main interest centres in the nature of the obstruction. It is a very vexed question as to what is the actual cause. In the case of adults it is referred to as cardiospasm, or some people prefer to call it achalasia—i.e., a spasmodic closure of the œsophagus without any obvious organic change being present in it. In the cases I have brought forward there is no visible pathological change, nor even any inflammatory condition, seen in the œsophagus itself; all that is seen with the œsophagoscope is the stricture when it occurs.

In one of these children the symptoms began when the child was 4½ years old, and she is now aged 11. She goes on quite comfortably except when the stricture



develops. It develops rapidly when it does occur, and equally it responds very rapidly to dilatation. When this difficulty in swallowing supervenes, she puts up with it for two or three days, and then she comes to hospital and is dilated with bougies. Dilatation is begun with rather a small size bougie and is worked up to almost the largest size, which the child takes quite well, and although it is uncomfortable she looks forward with such anticipation to her next meal that she endures it readily.

In the second case, that of a younger child, there was exactly the same condition,

44 Jewesbury : *Two Cases of Spasmodic Stricture of Œsophagus*

which only required four dilatations. The last attack occurred in June, 1923, and there has been no recurrence of the condition.

I looked up the cases of cardiospasm which we have had at St. Thomas's Hospital during the last three years, and I found that altogether there have been five. Of these, four were in women and one occurred in a man. Of the women, the age of the youngest was 30 and of the oldest 44. The man was aged 60. The treatment has been variable in these cases. Some have been treated with periodic dilatation, as in the case of these children. Only one of the patients was operated upon; the operation consisted in opening the stomach and dilating the strictured part of the œsophagus with the fingers. In the one case in which that was done the result seemed to be particularly satisfactory. The operation took place some time ago, and no spasm has occurred since. In one of these cases the patient was treated with belladonna, but I am not quite sure with what amount of success. Have other Members come across similar conditions, or any cases of dysphagia in children, and, if so, what are their views as to treatment?

Discussion.—Dr. W. M. FELDMAN asked whether Dr. Jewesbury had tried the injection of atropine in order to see whether the stricture disappeared.

Dr. F. PARKES WEBER suggested that in true "cardiospasm" or "achalasia of the cardia"—as distinct from hysterical and reflex œsophageal spasms—there was no permanent cure (unless possibly an operative one), either in children or adults. "Clinical cure" might last for years, but even in clinically successful cases, in children as in adults, X-ray examination might show that there was still more or less obstruction at the cardia and that the œsophagus above the cardia was dilated. Even after seventy years of age a patient might die from an exacerbation of the disease, that is to say, from cardiac syncope due to the chronic inanition. The rubber œsophageal tube with which such patients learned to feed themselves might coil up in the lower (dilated) portion of the œsophagus. The patient might not know that he was pouring food into his œsophagus and that he was "vomiting" from his œsophagus and not from his stomach.

With regard to children, there was an opportunity for the younger Members to try to find out what the prognosis was in these early cases. The cases would need following up for years. There was no doubt that proper treatment could bring about a satisfactory condition which might last for some years, but he (Dr. Weber) was inclined to think that one must never talk of a cure in these cases, because the trouble might begin again after some years' interval and even cause death (by chronic inanition and its results).

Mr. J. R. GRIFFITH, in discussing the pathology of the cases, said that some three or four months ago some beautiful sections were shown at a clinical meeting of the Society (demonstrating atrophy of the cells of the plexus of Meissner and Auerbach). He believed that they came from St. Thomas's Hospital. Did Dr. Jewesbury think that atrophy of the plexus was associated with the cause of the condition in these children?

Dr. JEWESBURY (in reply) said that atropine injections had not been tried. The response to dilatation had been so ready and had lasted for such considerable intervals that no other form of treatment had been tried up the present; but if there was any sign of the recurrence becoming more frequent that might very well be tried.

Dr. Parkes Weber threw some cold water on the prognosis, but what he (Dr. Weber) had said was probably correct. One of the cases he (the speaker) had mentioned, from St. Thomas's Hospital, was that of a man, aged 60, in whose case this diagnosis was given, and the history was that he had sudden difficulty in swallowing; the skiagram showed a shadow giving a series of rings or annular constrictions, with a big bag at the lower end, which apparently was always full. The height of that bag was about 8 in. Apparently there was a certain amount of leakage going on, and this had ceased when the man came up for treatment. In that case, apart from the dilatation and stricture the œsophagus showed no pathological change.

With regard to the destruction of the nervous mechanism mentioned by Mr. Griffith,

he was afraid that they could not take the credit for those important investigations at St. Thomas's Hospital; he rather fancied the work was done by Dr. Raikes at Guy's Hospital. Atrophy and degeneration of the cells of Auerbach's plexus had been demonstrated, and no doubt this would prove to be an important cause of some of these cases. Cases in which no obvious cause was present would probably be found to be due to the destruction of these nerve cells.

Birth Injury.

By W. FEDDE FEDDEN, M.S.

C. G., AGED 7 years.

November 9, 1920.—Patient first attended Victoria Hospital with mal-united fracture of left tibia and fibula just above the ankle-joint. Fractured at birth; normal presentation; long delivery. X-ray shows: Greenstick fracture of both bones just above ankle-joint; foot bent forward in extreme calcaneo-valgus position. (X-ray I, II.)

November 10, 1920.—Osteoclasis put in plaster; patient, aged nine months. (X-ray III.)

November 17, 1920.—Plaster reapplied. (X-ray IV.)

November 25, 1920.—Discharged; slight inward bowing of tibia. (X-ray V, VI, *a*, *b*, *c*.)

November 19, 1921.—Readmitted; cuneiform osteotomy; no tenotomy, put in plaster in equinovarus position. (X-ray VII, VIII, *a*, *b*.)

November 26, 1921.—Began walking, ordinary boots worn.

March, 1927.—Came up as patient, was not walking well. (X-ray IX, *a*, *b*.) Pelvis dropped. Scoliosis. Left leg appears perfectly sound; 1½ in. shortening left leg. Site of osteotomy hardly visible. One inch shortening of left arm noticed for first time.

I do not consider the case as one of birth injury, but of congenital deformity. The most striking feature is the congenital shortening and curvature of the diaphysis of the tibia with calcaneo-valgus. After osteotomy the diaphysial shortening has persisted. Although the leg has done extremely well, it is now discovered at the age of 7 years that the left arm is shorter by 1 in. than the right.

Discussion.—Mr. B. WHITCHURCH HOWELL said he thought the re-alignment of the tibia perfect. He thought the difference in the length of the arms was congenital and abnormal because it was beyond the usual difference in length in which the limb on the right side was longer than the left. Perhaps the abnormality in the tibia was due to lack of amniotic fluid in the uterus, causing abnormal pressure on the bone.

Mr. JENNINGS MARSHALL said that one had seen cases presumably of congenital shortening both in the arm and in the leg. He saw one recently at Charing Cross Hospital in which one limb was 3 in. shorter than the other, but in which no other pathological change could be found. The fact that they were congenital conditions had simply to be accepted.

Case of Congenital Absence of Femur, and Case of Effusion into Knee-joint.

By C. MAX PAGE, D.S.O., M.S.

IN the first of these cases there is a complete absence of the shaft of the left femur, the left leg being 8 in. shorter than the right. The epiphyses of the left femur are present, though relatively rather smaller than those of the opposite side. The thigh is represented by a distorted mass of muscle, over which the child has

46 Page: *Congenital Absence of Femur;* Firth: *Hyperkinesis*

some control. She can walk with distortion of the spine, but there is no stability in the leg. The main point to consider is whether one should do something now or wait until later. One might try, as a conservative measure, to fix the tibia into the acetabulum having first removed the epiphyses of the femur, and possibly at a later date remove the foot, leaving a thigh stump formed by the tibia. After the girl has grown up she could be fitted with a good artificial limb, with the knee-joint at the proper level.

The other case of interest is that of a girl with the rather obvious appearance of a congenital syphilitic who had an effusion into the left knee-joint. The X-rays showed the appearance of caries of the diaphysis of the femur just above the epiphyseal line, on the inner side. The Wassermann reaction was strongly positive. The child had been treated on anti-syphilitic lines for six weeks. The two knee-joints are now clinically normal; but one can still feel the thickening of the femur on the left side. A radiogram still shows the carious process in the bone. I think this is a syphilitic manifestation, and not due to tuberculosis.

Discussion.—Mr. B. WHITCHURCH HOWELL said he thought nothing should be done at present. When the child was 12 years old disarticulation should be performed at the hip-joint, and a tilting-table peg leg supplied.

Mr. JENNINGS MARSHALL said he thought that in the first of his cases the procedure proposed by Mr. Page was the likeliest to give the best results, namely, fixation of the tibia into the acetabulum and amputation of the foot. He (the speaker) would not, however, have this done at present. Several years might be allowed to pass before the operation was undertaken.

Dr. W. M. FELDMAN asked whether, in the first of these cases, it would be possible to transplant another bone, either from a recently deceased human being, or possibly from a living animal, so as to make a new femur. If this were done it would save the amputation of the foot.

Mr. MAX PAGE feared the suggestion made by Dr. Feldman was not applicable. It might be tried, but he would have no hope of a good result.

Case of Hyperkinesis of Doubtful Cause.

By DOUGLAS FIRTH, M.D. (shown by NEILL HOBHOUSE, M.B.).

E. F., GIRL, aged 2 years. History obscure; said to have developed normally, and to have sat up, walked, and begun to talk at the usual times. In March, 1927, lost use of limbs, and kept moving the hands aimlessly.

On admission there were incessant movements of hands in which both choreiform and athetoid elements were present. The child could not feed herself. Does not talk. There is no pyramidal lesion.

There is slight cardiac enlargement with a systolic murmur.

Dr. NEILL HOBHOUSE: There is some doubt about the heart condition; I have suggested congenital disease, chiefly owing to the early age and the absence of a rheumatic factor. As for the movements, I do not think it will prove to be a case of Sydenham's chorea; the condition might be one of natal or prenatal origin, or might be due to encephalitis. In any case I believe that it is due to a residual condition. The child was under two years of age when the movements began.

Dr. CHODAK GREGORY said she thought the case was most likely one of chorea. The reason why the movements were atypical was probably because the condition was in occurrence at a very early age. From an examination of the child she did not think that the aimless movements were confined to the hands.

The Use of Pedicled Skin Flaps in the Treatment of Syndactyly.

By C. JENNINGS MARSHALL, M.S.

A FEMALE child was brought with fusion of the second, third and fourth digits into a rounded mass. X-ray examination confirmed the absence of interphalangeal joints. It was, nevertheless, thought worth while to separate the fingers.

Clearly there would not be enough skin to provide a covering for all the fingers. In 1922 separation was effected: the whole of the available palmar skin was utilized to wrap round the middle digit, and the raw palmar surfaces of the other two were slipped under a cutaneous tunnel on the buttock, the flap being appropriately sutured proximally and distally. The skin was cut loose at the end of twelve days from its buttock attachments and adjusted.

In 1925 an Agnew type of operation was done for a small web at the interval between the index and middle fingers, and it is proposed to repeat this in the case of the middle and ring fingers.

At present the fingers possess good independent mobility, and the procedure appears justified from the æsthetic point of view also. The transplanted skin, which now has very nearly normal sensation, occupies relatively a much smaller area than at the time of transplantation.

Teratoma of Mesentery.

By C. JENNINGS MARSHALL, M.S.

PATIENT, a girl aged 8 years, was brought with a history of four days' abdominal pain. There had been occasional previous attacks. Temperature not elevated; there had been slight retching but no vomiting or anorexia. Tongue clean.

A rounded tumour was easily felt through the flaccid abdominal wall; it was remarkably mobile and could be placed in any part of the abdomen. A second smaller swelling could be felt, apparently attached to it by an elongated pedicle. Provisional diagnosis: Ovarian dermoid.

The tumour shown was resected with the segment of small bowel implicated. No other developmental defect was present in the abdomen. The mesentery was infiltrated with the thick fatty mass for a considerable area, the surface in places showing a warty appearance.

DESCRIPTION OF SPECIMENS.

An opaque walled cyst (contents at first fluid, but solidifying on cooling after removal from the body) contains fat. At one point it implicates the gut wall. Its interior is smooth-walled and shows no hair, bone, etc.

A larger thin-walled, translucent cyst, in which swirls of cholesterin flakes could be seen, affects the gut for a considerable distance. Its clear contents coagulated into a floccular slightly opaque mass under the action of the hardening fluid.

Section into the thickened mesentery at the widest part revealed widely differing

48 Marshall: *Teratoma of Mesentery; Use of Pedicle Flaps*

appearances. There are small, clear cysts, brown areas having a somewhat glandular look, cysts containing material closely resembling yolk of egg. No calcareous or bony material was found.

Section showed sebaceous glands, large colloid vesicles, involuntary muscle-fibres and areas of cells resembling adrenal cortex.

The nature of the tumour can hardly be in doubt. Its situation in the mesentery is of very considerable rarity; although it is recorded in Kaufmann's "Pathology" as occurring, no direct references to it have been found.

As regards pathogenesis, it may be supposed that some portion of the intermediate cell mass has been carried forwards in the dorsal mesentery.

Two Cases Illustrating use of Pedicle Flaps.

By C. JENNINGS MARSHALL, M.S.

Case I.—Child aged 1 year. Large cavernous angeioma of heel—had repeatedly ulcerated following application of CO_2 snow. The situation clearly precluded the use of Thiersch grafting or a local plaster. The angeioma was excised and immediately two-thirds of the area was covered by a pedicled buttock-flap, which fitted entirely without tension when the knee was flexed. Dressed with B.I.P. paste, windowed plaster applied. Child was nursed prone. Thanks to the skill of the ward sister, there was no infection throughout. The buttock pedicle was narrowed on the twelfth day by incisions on either side (under novocain). Flap completely divided on the eighteenth day, again under novocain, and the final adjustment on the heel made at the same time.

Note.—(1) The child submitted most patiently to the procedure, was at no time peevish and took food normally.

(2) The very short time taken for the operation. This was absolutely adequate, there being numerous arterial spurts from the heel side of the final dividing incision.

Case II.—Boy, aged 7 years. Very large hairy and papillomatous mole of right forehead, root of nose, malar region, and temporal region. CO_2 snow and radium applied in Australia.

As time is of no very great object at this age, in view of the great disfigurement the operation of excision and replacement by a pedicle flap is proposed. A neck pedicle with a pectoral flap was rejected on account of the extra visible scarring. It is proposed to transfer an abdominal skin flap, the forearm being used as the intermediate site. The child is shown with the abdominal flap attached to the forearm a month after the first suture of the pedicle to the limb. The amount of skin will need further increase by extension of the incisions before the necessary large area—about 4 in. by 3 in.—is available.

Note.—(1) A much longer period appears to be necessary than in the preceding case, the parts concerned being much less vascular. The boy does not seem at all worried by the constraint; I think the mental effect of protracted and repeated procedures has, however, to be taken into account.

(2) The possibility of considerable fatty development in the transplant has to be borne in mind. Although this lad was of slim build (and so also were his parents), it is difficult to predict what degree of abdominal subcutaneous fat may be present by middle age.

Mr. W. FEDDE FEDDEN said he considered that with web fingers it was not possible to get a satisfactory result from the ordinary text-book operation; he thought the pedicle-flap was of great value in such cases.

Section for the Study of Disease in Children 49

Spastic Diplegia.

By NEILL HOBHOUSE, M.B.

J. H., GIRL, aged 4½ years. Delivery normal. Has never walked. Did not talk until 3 years. Always held left arm stiff.

Signs on Admission last February (1927).—Moderate spastic weakness of right arm, which is not used. Supination limited. Abdominals absent. Considerable spasm of legs. Deep reflexes. Plantars extensor.

Treatment.—The sound arm was bandaged to the trunk, while the child was in bed, and active movement in the spastic arm rapidly increased. The child was taught to walk in a wheeled supporting frame, and after a week or so could push a doll's perambulator. She was, however, unable to get the right heel on the ground owing to active spasm of the calf.

On April 8 Mr. Jennings Marshall exposed the nerve to the soleus muscle and injected it with absolute alcohol. It is hoped that the child may be able to walk alone by the time that power returns in this muscle, function being maintained in the gastrocnemius.

A Case of Acute Lymphatic Leukæmia.

By NOEL OLIVIER RICHARDS, M.D.

BOY, aged 9 years.

History.—"Influenza" during first two weeks of February, 1927. In bed fourteen days for cough and sore throat. After getting up was very easily tired, nervous and depressed, and very pale. There was a slight attack of nose-bleeding four weeks after the onset of the illness.

Previous History.—Severe epistaxis after measles three years ago, and again in December, 1926.

Family History.—Two other boys aged 7 years and 4½ years alive and well. Father has "D.A.H."

Symptoms on March 16, 1927.—Pallor, fatigue, nervousness, depression, slight cough.

Physical Signs (March 15).—Rather thin, very pale, yellowish complexion with pale mucous membranes. The superficial veins of the neck and upper part of chest anteriorly were prominent (no œdema, no hæmorrhages). *Heart* enlarged, epigastric pulsation visible. Apex beat in sixth space one inch beyond mid-clavicular line. Sounds rapid, with gallop rhythm, clear at apex, muffled at base. Systolic murmur at apex, conducted to axilla. *Lungs.*—Tubular breath sounds at the angle of left scapula. *Liver* enlarged. Margin one finger's breadth below costal margin. *Spleen* enlarged, the edge extended two fingers' breadth below costal margin, rounded and firm, no notch felt. *Tonsils* enlarged. *Lymphatic glands* of neck, axillæ and inguinal region palpable, firm and discrete. No large mass, but definite enlargement of the units in every group.

Blood-count (March 27, 1927, by Dr. Wyard).—Total red cells, 1,390,000 per c.mm. Hæmoglobin, 26 per cent; colour index, 0·9; total white cells, 1,080,000 per c.mm. No differential count is practicable. Now and then a polymorphonuclear

leucocyte or an erythroblast is seen, but for the most part the film consists of a close mass of small lymphocytes.

Treatment.—This was at first directed towards the relief of the cardiac condition. On March 29 administration of liquor arsenicalis was begun and has been continued since. Iron has also been given since April 9.

Progress.—On April 10 the heart was found less enlarged. The heart sounds had become clear at the base. The liver was smaller and the veins less engorged. The spleen appeared to have increased in size; the hæmoglobin was 20 per cent.

Discussion.—Dr. STANLEY WYARD said he did not remember having seen a case in which such a large number of leucocytes were present, the leucocytes and the red cells being practically equal in numbers. The films showed very few polymorphonuclear cells, and very few properly formed lymphocytes. The majority of the white cells were myelocytes, with a proportion of myeloblasts. The usual changes were taking place in the red cells, and there were a certain number of erythroblasts. The condition as shown by that blood-picture was very acute. The immature white cells indicated that the process was very rapid; in his opinion the prognosis was very grave. Probably two or three months would see the end of the patient.

Dr. RICHARDS (in reply to a Member of the Section who asked whether the child had been subjected to artificial sunlight or whether the bronze colour was incidental to the disease), said that no artificial sunlight treatment had been given.

POSTSCRIPT.

I believe the usual course of these acute cases is two or three months. The illness was first noticed at the beginning of February, 1927, in this case. Now (June 26), the patient is not expected to live for more than *a few weeks* longer. N.B.—The anæmia probably began before the child was thought to be ill. He had epistaxis in December, 1926.

Petit's Eventratio Diaphragmatica.

By C. BLAXLAND LEVICK, M.B.

I. T., AGED 5 years, female. For seven weeks: wasting and failure of appetite, dry cough with occasional vomiting. "Congestion of left lung" at three months. No other serious illness. When the asymmetry of the chest was pointed out to the mother, she declined to agree that any deformity was present and said that the child "had always been like that."

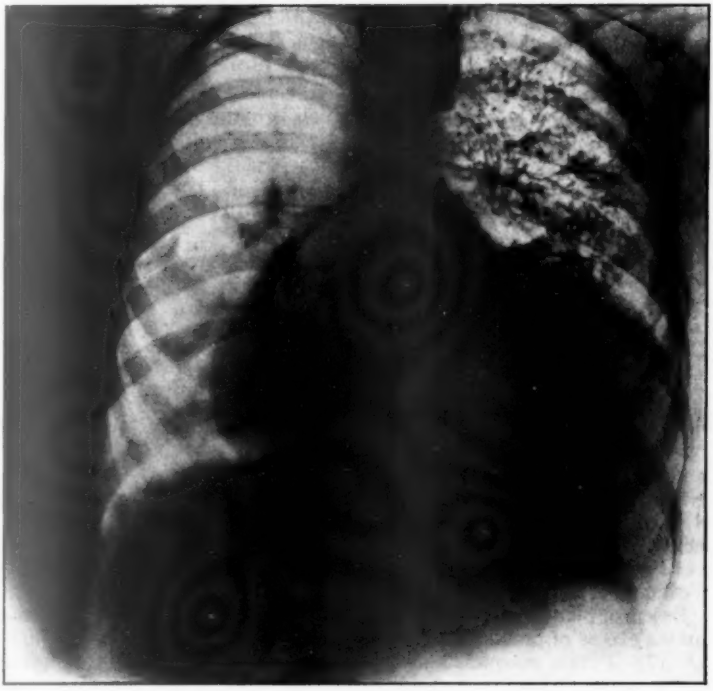
Family History.—Only child. Paternal grandfather and two paternal aunts are said to have suffered from tuberculosis.

On Examination.—Pale and spare; throat clean; tonsils small. Slight scoliosis, convexity to the right. *Heart*: Apex-beat in fifth space just outside the nipple line; otherwise normal. *Chest:* Marked asymmetry, left side uniformly larger in front, perhaps entirely due to the scoliosis. *Right base:* Generally retracted especially anteriorly and laterally, and to a greater extent than can be attributed to the scoliosis. Slight indrawing of lower chest-wall on inspiration. Vocal fremitus practically absent. Percussion note dull but not stony dull. Breath sounds absent over an area at the base of the right axilla which is surrounded by distant vesicular breathing. No

adventitious sounds. Vocal resonance diminished. Physical examination otherwise negative.

X-ray Examination (Dr. R. L. Rawlinson): "Some scoliosis, the heart lying to the left in the concavity of the curve. Right side of diaphragm is very high and almost immobile, ? paradoxical in movement, suggesting phrenic paralysis. Ill-defined opacity proceeding from the root of the right lung (? neoplasm), which appears to be anteriorly placed in the lateral view."

During a fortnight in hospital the general condition improved and the weight



increased. Temperature was irregular, 98° to 100° F. An exploratory needle failed to find fluid or pus.

Lipiodol, 15 c.c., was injected through the crico-thyroid membrane, and a further X-ray showed "the right side of the diaphragm very high in position and moving poorly and paradoxically. Though the descending bronchus is patent, the lipiodol does not flow down to the base of the lung as far as usual but is visible through the right dome."

This case illustrates a congenital condition in which one half of the diaphragm is a thin fibrous structure, practically devoid of muscular tissue. It therefore extends

high into the thorax; having no power of contraction it is subject to the mechanical forces of respiration resulting in "paradoxical respiratory movement," i.e., upward in inspiration and downward in expiration. It has been ascribed to a failure of development or to phrenic paralysis at birth.

The condition may be clinically and radiologically indistinguishable from unilateral phrenic paralysis arising after birth; but the diaphragm in the latter case forms a thicker line, and some obvious pathological cause is to be expected, or else a history of pneumonia, or of diphtheria, on the same side.

The condition is not uncommon on the left side. The present case, however appears to be the first of right-sided eventration to be recorded in this country; it is practically identical, clinically and radiologically, with one described by Bayne Jones [2] in which the diagnosis was confirmed at operation. The one important difference consists in the deformity of the chest wall in the present case. This deformity, however, seems to occur only in cases associated with imperfect development of the lower lobe of the lung, which is here seen in the lipiodol X-ray.

REFERENCES.

[1] J. Woodburn Morison, *Arch. Radiol.*, 1923, 353. [2] S. Bayne Jones, *Arch. Int. Med.*, 1916, 221. [3] H. M. Korns, *ibid.*, 1921, p. 192.

Tuberculoma of Cerebellum.

By Stanley Wyard, M.D.

C. G., aged 8 years.

Cervical glands enlarged since June, 1924. On March 4, 1927, they were dissected out. Some were caseous.

March 5.—Temperature 102° F.; complained of headache.

March 6.—Temperature and headache unchanged. Pain also in back of neck. Restless and constipated.

March 11.—Mental state very dull. Takes no notice of surroundings. Is very irritable and resents interference. Some photophobia. No strabismus. Rigidity of neck. Weakness of left upper and lower limbs. Deep reflexes brisk. All abdominals absent. Extensor plantar response both sides. Kernig's sign marked. Cerebro-spinal fluid contains excess of protein and a marked lymphocytosis, but there is no clot.

March 17.—Pupils unequal, and there appears some weakness of left external rectus. The right abdominal reflexes have returned. The right plantar reflex is indefinite, the left remains extensor. At no time has there been any change in the ocular fundi.

March 25.—Mental condition improving; he now notices his surroundings and obeys instructions. There is dysdiadokokinesis, but no ataxy. Weakness of the left limbs persists. The abdominal reflexes are reappearing. The left plantar reflex is flexor, the left extensor.

During April he has made steady progress. His mental condition is becoming more nearly normal, and the disability is diminishing.

This is rather an interesting case from the point of view of diagnosis and of determining what exactly has happened. The child had definite tuberculous glands in the neck which had existed for some considerable time; some of these were eventually removed by operation. On the following day the temperature rose, and the boy complained of headache. The disease rapidly progressed and the mental

condition showed typical features of cerebral irritation—the boy lay curled up in bed, he resisted all examination, there was some degree of photophobia, there was a certain amount of stiffness of the neck, and Kernig's sign was positive. But at the same time he showed definite changes in the reflexes. All four abdominal reflexes were lost, and there was extensor response on both sides. The cerebro-spinal fluid was faintly opalescent. Microscopically, it was found that there was marked lymphocytosis. I was fairly confident of the diagnosis of meningeal tuberculosis and made a prognosis that the child could not possibly recover. But fortunately the child made definite improvement almost at once, and his condition is now very different from what it was formerly. When alone he sits up and plays with his toys and takes intelligent interest in his surroundings. As he progressed, the abdominal reflexes returned, first on the right side, and then on the left. The plantar reflexes altered in a similar manner, first the right side becoming flexor, and then the left side. After the mental condition began to improve it was obvious that he was weak down the left side—the upper limb and the lower limb were definitely weak. Dysdiadockokinesis was obtainable, though not quite so obvious as it often is in cases of cerebral tumour. There has been no ocular change at any time during the illness.

Discussion.—Dr. J. D. ROLLESTON (President) asked whether a bacteriological examination was made of the cerebrospinal fluid. He thought this was one of the cases corresponding to those described by French writers as "curable meningeal conditions," that is to say, conditions in which the clinical picture resembled that of tuberculous meningitis, but differed from the great majority of cases of tuberculous meningitis in that recovery took place. Of course, there were a fair number of cases of apparently genuine tuberculous meningitis which had ended in recovery, even where the clinical picture was quite definite, but there were a larger number of cases in which the clinical picture was not so complete but in which the patients had finally recovered.

Dr. WYARD (in reply to the President) said that he did examine the fluid bacteriologically, and was unable to find any tubercle bacilli or other organisms.

Case of Anæmia.

By R. C. JEWESBURY, M.D.

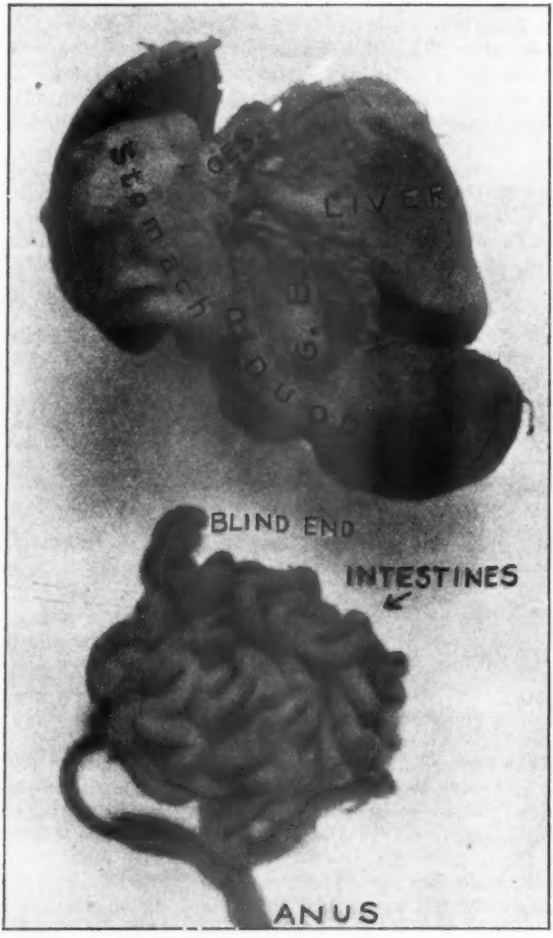
I HAVE brought forward another case which has one or two points of interest, that of a child, just over two years of age, who was brought here for nothing very definite. The history was that the patient did not seem to be getting on well. He was a very rickety child, and had a very large spleen and also some enlargement of liver. The point is the blood examination. Dr. Wyard reported that the blood contained 1,200,000 red cells per c.mm., and just over 8,000 white cells. There was nothing particular in the differential count, and there were six myelocytes and two myeloblasts present in the count. The diagnosis lies between a splenic anæmia, a leukæmia in a very early stage or pseudo-leukæmic anæmia of the von Jaksch type. I am of opinion that this latter condition is the most likely.

Dr. STANLEY WYARD agreed with Dr. Jewesbury's deduction. It corresponded to his own idea following upon the blood-picture. The second blood-picture showed a considerable number of erythroblasts as well.

54 Myers: *Atresia of the Duodenal Junction*

Atresia of the Duodenal Junction.[1]

By BERNARD MYERS, C.M.G., M.D.



Specimen showing (*a*) distended stomach, distended duodenum, the latter containing bile-stained fluid; liver and gall-bladder normal; (*b*) blind end of the duodenum, intestines, and anus.

[1] This illustration was inadvertently omitted from the last number of the *Proceedings*. For description of the case and specimen, see *Proceedings*, 1927, xx (Sect. Study Dis. Child.), p. 28.

Clinical Section.

President—Sir HERBERT WATERHOUSE, F.R.C.S.

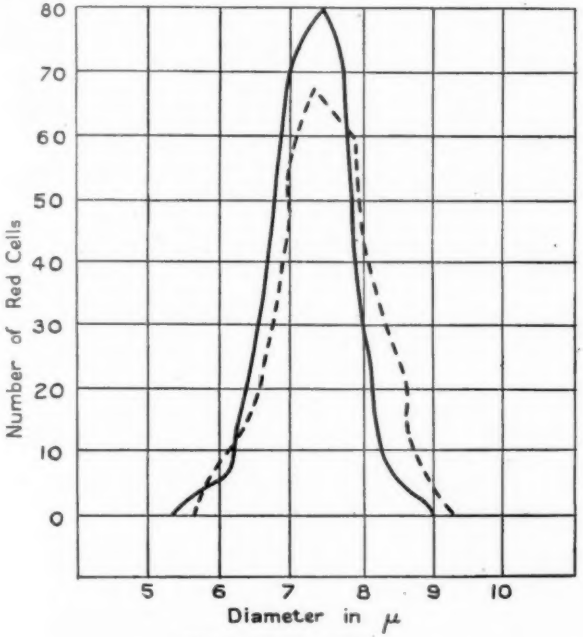
Combined Degeneration of Spinal Cord.

By M. A. CASSIDY, M.D.

C. J., AGED 64, a bricklayer.

History.—Early in December, 1924, he noticed pallor; saw a doctor who said his complexion was so yellow that he suspected jaundice. Teeth were very septic; all were removed, and large doses of arsenic were administered (up to 12 minims t.d.s.). Rapid improvement followed; but about the middle of January, 1925, he noticed that his hands and feet were weak and stiff, and had a feeling of pins and needles. Less than a fortnight after this he became so ataxic as to be quite unable to walk, or even to stand, unsupported, and his hands were also useless; he could neither write nor dress himself. At this time there was also a feeling of constriction about the level of the umbilicus. He had noticed that his tongue was tender at times. His bowels were constipated throughout; there was never either diarrhœa or vomiting.

On admission to St. Thomas's Hospital on February 2, 1925, there was no apparent loss of power nor any sensory defect, but he was still wildly ataxic and unable to stand or to button his clothes. Knee-jerks absent; plantar reflexes flexor; abdominal reflexes brisk. Loss of vibration sense in left leg and left side of pelvis.



EXAMINATION OF BLOOD.

	Feb. 9, 1925	Mar. 20, 1925	Aug. 12, 1925	Jan. 13, 1926	Nov. 23, 1926	Jan. 5, 1927
Erythrocytes	4,820,000	5,300,000	4,300,000	4,520,000	3,320,000	4,380,000
Hæmoglobin	94	110	92	92	90	90
Colour index	0·98	1·04	1·05	1·01	1·35	1·04
Leucocytes	4,400	2,800	5,000	2,520	—	2,640

STAINED BLOOD.

	Feb. 9, 1925	Mar. 20, 1925	Aug. 12, 1925	Jan. 13, 1926	Nov. 23, 1926	Jan. 5, 1927
Polynuclear neutrophils ...	53·5 ...	50·5 ...	41 ...	47·5 ...	— ...	73
,, eosinophils ...	3 ...	0·5 ...	2 ...	3·5 ...	— ...	—
Small lymphocytes	30 ...	29·5 ...	44 ...	17·5 ...	— ...	13
Large ,, ...	8 ...	15 ...	6 ...	21·5 ...	— ...	10
Large hyaline cells	4 ...	4 ...	6 ...	10 ...	— ...	4
Coarsely granular basophilic cells	1·5 ...	0·5 ...	1 ...	— ...	— ...	—

No microblasts, normoblasts, or megaloblasts in any of these counts, with the exception of that of January 5, 1927, when one megaloblast was seen.

Pryce-Jones curve lower and wider than normal—average diameter red cells 7·56 microns (normal 7·23 microns).

Van den Bergh's reaction, direct and indirect, both negative.

Test meal, February 5, 1925.—No free hydrochloric acid ; no lactic acid.

Cultures from duodenal contents showed an abundant growth of non-hæmolytic cocci, but only one or two colonies of hæmolytic streptococcus.

Wassermann and Sachs-Georgi reactions negative in blood and cerebro-spinal fluid.

Treatment.—Educational exercises. Dilute hydrochloric acid in drachm doses with meals. Dimol ; occasional courses of arsenic.

Progress.—Slow progressive improvement ; by December, 1925, he was able to walk with the help of a stick, and now he can just manage to walk without a stick and can write and dress himself. Recently a definite Babinski's sign has appeared on the right side.

When he first came under observation some physicians considered the case to be one of anæmia secondary to dental sepsis, cured by treatment, but complicated by an arsenical neuritis. Subsequent events seem to confirm the original diagnosis of pernicious anæmia with combined degeneration of the spinal cord.

May 13, 1927.

Gumma of Liver, Simulating a Perforated Gastric Ulcer.

By PHILIP TURNER, M.S.

A. W. K., MALE, aged 24, was admitted for acute abdominal pain.

History.—For six years patient had suffered from abdominal pain, particularly in the right iliac and hypochondriac regions, coming on after taking food.

On the day of admission he was seized with acute pain in the epigastric region, associated with marked tenderness and extreme rigidity of the upper part of the abdomen above the umbilicus. Tenderness and rigidity were completely absent in the lower part of the abdomen. The pulse, temperature, and respiration were normal. A perforation of a gastric ulcer, with a localized abscess, was suspected ; but in view of the normal pulse-rate and the absence of any tenderness and rigidity of the lower abdomen it was decided to keep the patient under observation. The pulse-rate remained normal and the pain and tenderness gradually diminished in the course of the next two or three days. As this happened it became obvious that there was a tumour present in the epigastric region which came down from beneath the ribs and was resonant on percussion. It was thought that a gastric ulcer had leaked into the lesser sac, and five days after admission a laparotomy was performed. Upon the abdomen being opened the stomach was found to be normal, but there was a large tumour of the liver just to the left of the falciform ligament and a second smaller tumour was present on the right side of the neck of the gall bladder. The tumour was thought to be a neoplasm, and after an incision had been made into it a portion was removed for histological examination. This showed that the tumour was a gumma. The patient then gave a history of syphilis contracted ten years ago and the Wassermann reaction was strongly positive. He is being treated with injections of N.A.B., with the result that the tumour has greatly diminished in size.

Fractured Patella treated by Wiring in 1889.

By PHILIP TURNER, M.S.

S. G., FEMALE, aged 60, was admitted for a painful hammer-toe, which was treated by amputation.

It was found that thirty-eight years ago, in 1889, she had been admitted under the late Sir Henry Howse for a fracture of the left patella, which was treated by wiring. A single wire was used which did not penetrate the articular surface. The X-ray plate shows the wire still in position. There is perfect bony union, the outline of the patella is normal, and there is no irregularity of the articular surface. A slight degree of arthritis is present, but not more than would be expected in a patient of this age.

The case is shown as the end-result of a very early case of a fracture of the patella treated by wiring. The result can only be described as perfect. There is full movement of the knee-joint, the joint has been painless and there has been perfect function since the operation. There is a slight degree of grating on movement, but this causes no pain and a similar condition is present in the other knee.

Facial Hemiatrophy and Persistent Hiccough.

By C. F. T. EAST, M.D.

PATIENT, female, aged 34. The facial hemiatrophy first began about the age of 14. There are now well-marked changes in all the tissues of the right side of the

62 East—Slesinger—Worster-Drought

face and head. The tongue is small on the right side, and the opening of Stenson's duct is not apparent. The temporal area is anæsthetic. The hiccough has been persistent about thirteen years. It is brought on by emotion and exertion, and seems to be in the nature of a diaphragmatic tic. Sometimes she is free from it for long periods. She has improved a good deal with the practice of breathing exercises.

Arterio-venous Aneurysm with Bullet retained in Heart Muscle.

By E. G. SLESINGER, O.B.E., M.S.

R. W., MALE, aged 32, was wounded in the right shoulder in 1918. I first saw him in 1919 for an arterio-venous aneurysm of the right subclavian, his only complaint of which was, and still is, the noise, which often keeps him awake at night. He now complains of pain over the heart at times and shortness of breath. A radiogram shows the bullet to be in the heart muscle, probably in the front of the interventricular septum. An electrocardiogram by Dr. Campbell is not abnormal. There is an inverted T in lead III, and the P. R. interval is one-fifth of a second.

The pulse pressure in the right arm is only 10 mm. (right arm 65/55; left arm 115/55), but he has no trouble with the arm or hand.

The case is shown in order to consider the advisability of removing the bullet from the heart, in view of the pain and the risk of the bullet moving.

Spastic Paresis of One Leg.

By C. C. WORSTER-DROUGHT, M.D.

S. T. B., MALE, aged 40. Complained of weakness of the right leg and consequent difficulty in walking.

History.—In 1919 (aged 32) from January to June he complained of pain in the small of the back and both legs; the condition was diagnosed as "myalgia" and had completely disappeared by August, 1919. One morning during September of the same year, however, he found he was unable to use his legs properly; he had been quite well on the day before. He improved for a year or so, but since 1920, when the patient was first seen, the condition has remained stationary.

Physical Signs.—Gait with right leg somewhat spastic and foot inverted. Pupils, optic discs, and all other cranial nerves normal; no nystagmus. Arm muscles and reflexes normal; no tremors. Abdominal reflexes: brisk in the upper quadrant on both sides, sluggish in the left lower quadrant and absent in the right lower quadrant. Cremasteric reflexes: right absent, left sluggish. Right leg somewhat spastic with slight general muscular atrophy and weakness. Right knee- and ankle-jerks exaggerated and greater than those on the left; right patellar clonus and right ankle clonus. Plantar reflexes: right extensor; left usually flexor when the inner side of the sole is stimulated, extensor when the outer side is stimulated. Sensation everywhere normal. Coördination normal.

X-ray of spine negative.

Wassermann reaction negative in both blood and cerebro-spinal fluid; the latter also shows no abnormality.

Commentary.

The upper neurone paresis involves chiefly the right leg, the left being scarcely affected. The fact that the right abdominal reflex is absent in the lower quadrant enables one to localize the lesion at about the level of the tenth thoracic-spinal segment. Since no sensory changes can be detected, the lesion is confined to the white matter of

the cord. The relative suddenness of the onset, as well as the fact that after an initial improvement the condition has remained stationary since 1920, strongly suggests a vascular lesion—hæmorrhage or thrombosis. It is difficult, however, to suggest any cause for any vascular lesion in a patient of his age and in such a situation. Also, in most cases of hæmatomyelia the grey matter is very definitely involved and usually to a greater extent than the white matter.

The case is shown for any further suggestions as to diagnosis.

Dr. F. PARKES WEBER said he thought that such cases might sometimes be accounted for by a local thrombotic lesion in the spinal cord, arising in connexion with a temporary infection of some kind. In temporary infections (including those from *Bacillus coli* and clinically "influenza-like" attacks) there was probably a true bacteriæmic (septicæmic) stage during which local, non-suppurative, inflammatory lesions might occur in various parts of the circulatory system, including the brain and the spinal cord.

Case for Diagnosis.

By C. LAMBRINUDI, F.R.C.S.

E. A., FEMALE, aged 16. Eldest of six children. All the others are healthy. No history of any nervous or crippling disease in any members of the family. The earlier notes of her case cannot be found and her mother's account is unreliable, but it appears that when aged 2½ she fell down a flight of stairs; later she developed diarrhœa and vomiting, and pain in her back and neck. She was admitted into the medical wards at Guy's Hospital. Lumbar puncture was negative.

Her first records in the Orthopædic Department show that she was treated in plaster of Paris applied to both legs for about two years from 1913 to 1915 and from that time onward has constantly attended for remedial exercises.

She now walks with an extremely unsightly and awkward gait. Her knees and hips are a little flexed and her back markedly lordosed. Her knee-jerks are absent. The extensors of her knees, the psoas and adductors of her thighs are paralysed on both sides, and she has some contracture of her knees and hips. The gluteus maximus muscles are extraordinarily enlarged and firm and out of all proportion to its strength. She has a fibrous mass in the upper part of her left peronei muscles.

Is this a case of anterior poliomyelitis or an unusual form of pseudo-hypertrophic muscular dystrophy?

Dr. F. PARKES WEBER thought that the difference in size of muscles between the lower limbs was probably the result of early infantile paralysis, but in addition there might be a pseudo-hypertrophic form of primary muscular dystrophy in progress.[1]

Aneurysm of Innominate Artery; Spontaneous Disappearance of Swelling in Neck.

By DAVID KRESTIN, M.D.

PATIENT, J. G., male, aged 49, an interpreter, first came under the care of Dr. Theodore Thompson in August, 1926.

History.—He enjoyed good health until *seven months* before coming under observation (i.e., February, 1926). He then began to get agonizing pain in the back

[1] On nervous diseases supervening in the subjects of old infantile paralysis compare F. Parkes Weber, *Journ. Neurol. and Psychopath.*, London, 1923, iv, 40. In some subjects of infantile paralysis there seems to be a special liability to spinal cord disease. "But whether this liability in any cases extends to the motor nerves or their continuations—the muscles themselves—future observations must show."

64 Krestin: *Aneurysm of Innominate Artery*

of the head and neck. *Four months* later, this diminished, but similar pain occurred in the right shoulder and axilla. At the same time the upper part of the anterior chest wall on the right side and the right arm appeared swollen, and prominent superficial veins in this region were noticed by the patient. Six weeks after this, a swelling about the size of a damson appeared on the right side just above the clavicle and to the right of the sterno-mastoid. Though uncomfortable it was not painful. At the same time he lost his voice, felt short of breath on exertion, and cough commenced.

One week before admission to hospital on August 23, 1926, the swelling rapidly increased in size and became painful.

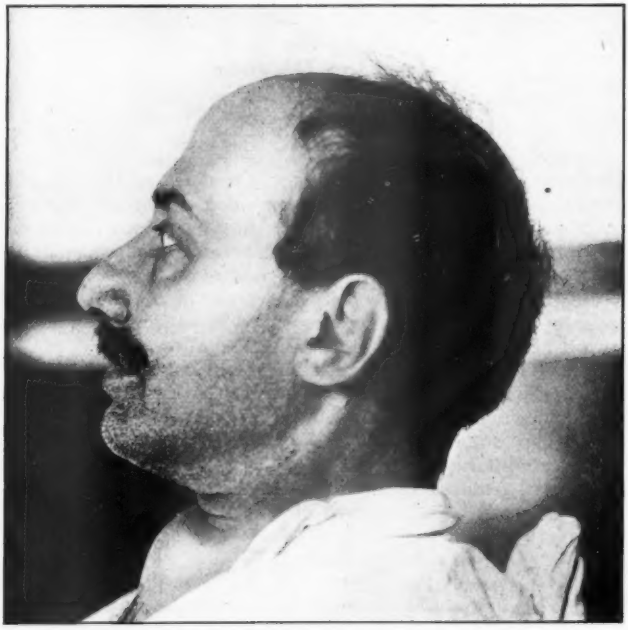


FIG. 1.

Previous History.—Twenty years ago he contracted gonorrhœa and a "soft chancre"; the latter was treated by cauterization. His right eye was injured during childhood. Has been married nine years. There were no children and his wife never had a miscarriage.

Examination in August, 1926.—Patient, a spare, muscular man with a staphyloma of the right eye. He had considerable dyspnœa, and inspiratory stridor was well heard. Appearing on the right side from behind the inner end of the clavicle and lower end of the sterno-mastoid, was a soft, uniform rounded swelling (fig. 1) about the size of a tennis ball, which showed expansile pulsation, was tender on palpation, and pushed the trachea to the left. No thrill could be felt, and on

auscultation over it the heart sounds and tracheal breath sounds were transmitted. No murmurs could be heard. The inner end of the right clavicle was pushed forwards. The heart was neither enlarged nor displaced; the apex beat was distinctly felt in the fifth space just inside the nipple line. Systolic pulsation, synchronous with that of the swelling, could be seen and felt over the manubrium sterni and for three or four inches to its right. A diastolic shock was felt and the

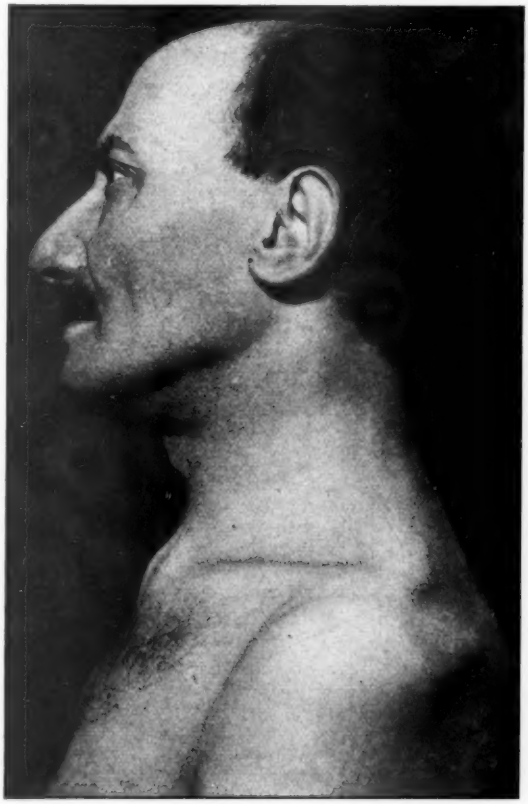


FIG. 2.

percussion note impaired over the same area. The cardiac sounds were clearly heard in all areas, the second aortic being accentuated. No murmurs were heard. Enlarged superficial veins were present beneath the right clavicle and in the right arm. Both pulses were equal, regular, and synchronous. The blood-pressure in either brachial was 135 mm. Hg systolic, and 90 mm. Hg diastolic.

Nothing abnormal was found on examination of the lungs and abdomen. A zone of hyperæsthesia was present on the right side of the neck, corresponding to C 2 and

66 Krestin: *Aneurysm of Innominate Artery*

C 3 spinal segments. There was abductor paralysis of the right vocal cord. The Wassermann reaction in the blood was positive.

The patient refused any form of treatment and went home.

Within three months of his departure, the swelling and pain had completely disappeared, his voice had returned, and his general condition improved, without any treatment at any time. He has remained at his work since.

Now, but for an indefinite thickening felt deep in the root of the neck, there is no sign of a swelling (fig. 2.) The sternal end of the right clavicle is still

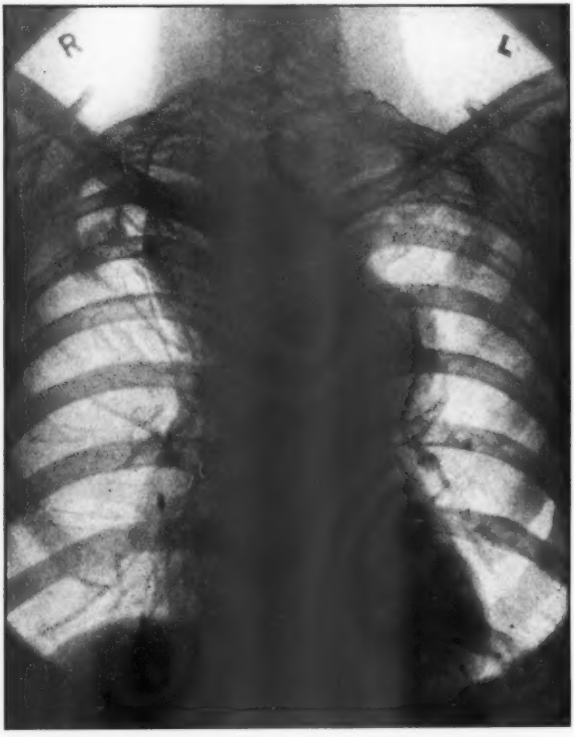


FIG. 3.

unduly prominent. His voice is quite normal and both vocal cords move well. Obvious pulsation is present in the right common carotid and superficial temporal arteries; both pulses are equal and synchronous, the blood-pressure in each brachial being 125 mm. Hg systolic, and 90 mm. Hg diastolic. Except for the absence of enlarged superficial veins over the chest wall, the other physical signs are unaltered. He can now walk two miles in comfort. The radiogram of the chest (fig. 3) shows dilatation of the ascending arch of the aorta extending upwards into the innominate artery.

Diagnosis and Remarks.—There can be no doubt that this man has an aneurysm of the ascending arch of the aorta extending up into the innominate artery. The condition is due to syphilis; there was never any history of trauma. The very rapid increase in size of the swelling almost within a week, and its remarkable disappearance, require explanation. It is suggested that a rather acute destructive process occurred in the wall of the vessel, and that blood leaked out into its sheath, so producing a false aneurysm. This may have followed some minor strain or effort of which the patient was unaware. Subsequently, most of this blood was absorbed and the remainder, after clotting, became organized. As can be seen from the radiogram, the main aneurysmal dilatation of the innominate artery and aorta is still present.

I am indebted to Dr. Theodore Thompson for permission to show this case.

Exophthalmos Cured by Carotid Ligation.

By St. J. D. Buxton, F.R.C.S.

Mrs. M., aged 45, was knocked down in the street on September 13, 1926. She was brought to the casualty department at hospital, detained a few hours and then sent home, no sign of gross injury being found.

Fig. 1.—Before operation.

68 Buxton: *Exophthalmos Cured by Carotid Ligation*

In October, she complained of diplopia, and was given glasses, after attendance at an ophthalmic hospital.

In January, 1927, she had pain in the right side of the head and was referred from the eye hospital.

In February examination showed that the left eye was proptosed and pulsating slightly. The conjunctiva was œdematous and overhanging the lower eyelid. There was only slight movement of the left eye and she could read nothing. She was able to count fingers in front of the eye. A loud murmur could be heard with a

FIG. 2.—After operation.

stethoscope when the latter was placed above the orbit. It was heard loudest when placed over the left frontal sinus. This murmur disappeared when pressure was made upon the common carotid artery.

The first photograph was taken on February 14.

On February 16 operation was performed. The three carotid arteries were exposed. It was found that the murmur disappeared when either the common carotid or the internal carotid were occluded on the left side, but pressure on the external carotid had no effect on the murmur. The left internal carotid was ligated.

The proptosis gradually went down and movements of the eye and vision returned to normal. The murmur never returned. She complained of feeling giddy in the morning for a few days.

At the present time she states that there is no trouble with the eye at all.

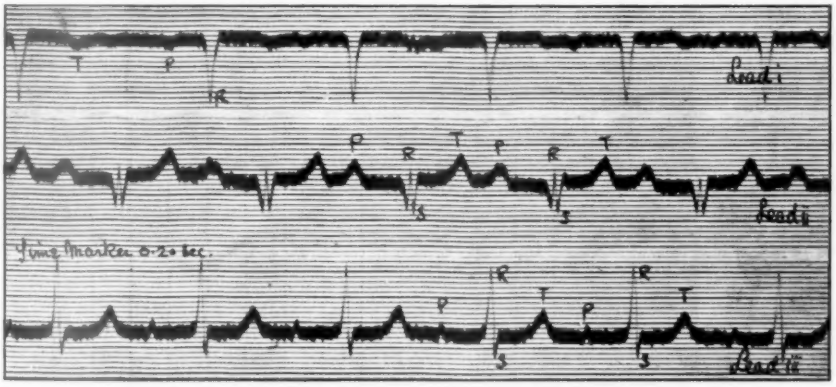
Complete Transposition of Viscera with Cardiac Complications.

By B. T. PARSONS-SMITH, M.D.

H. W. C., MALE, aged 19, has complained during the past winter of cough, breathlessness and occasional giddiness.

Previous History.—Growing pains and tonsillitis in childhood, influenza in 1926 and acute fibrinous pleurisy in February of this year.

Physical Signs.—Heart dullness mainly exactly to the right of the sternum, the maximum impulse being situated in the fourth right space 3 in. from the mid-line; rhythm regular except for an occasional premature contraction; diastolic shock and



diastolic murmur over the base of the heart, the murmur being conducted down the right sternal margin to the region of the apex impulse; pulse 84, fair volume, occasional intermissions; vessels normal for age; blood-pressure 130 mm. systolic, 60 mm. diastolic; fingers clubbed; spleen not felt; electrocardiogram typical of dextrocardia (all the waves in lead I being inverted) and partial heart block.

X-ray examination shows complete dextrocardia with transposition of the viscera. Blood-count normal; Wassermann reaction negative.

70 Burrell: *Carcinoma of Lung;* Hodson: *Amœbic Hepatic Abscess*

Early Carcinoma of Lung.

By L. S. T. Burrell, M.D.

Male, aged 56. Winter bronchitis for six years.

Hæmoptysis in January and several small attacks since.

Brown sputum. No tubercle bacilli found. General emphysema. Impaired note and weak breath sounds at apex of right lung.

X-ray shows an opacity with ill-defined margin in the upper part of right lung.

Case of Amœbic Hepatic Abscess, treated as an Out-patient with Emetine Only.

By V. S. Hodson, M.V.O., M.D.

H. K., aged 39. In June, 1926, he had dysentery in India and was successfully treated with emetine. He returned to England late in the year and began to feel ill in January, 1927. This illness was very indefinite and only slowly increased.

Fig. 1.

He came under my care on April 1. His condition was then very typical. He looked ill and had the muddy complexion which is so common in liver conditions. The temperature was normal at the time of examination. Physical examination showed limitation of movement of the right side of the chest with slight

prominence over the liver area. The percussion note was impaired below the angle of the scapula and liver dullness began in front in the fourth intercostal space in the nipple line. Breath sounds were deficient, particularly behind, and a few fine crepitations were heard but no bronchial breathing; voice sounds were not markedly altered. The X-ray picture taken that afternoon (fig. 1) shows a very definite upward enlargement of the right lobe of the liver with fixation of the diaphragm.

The patient was ordered emetine gr. 1 for three days and told to return in a week. It was taken for granted that, as he was under the care of his panel doctor, he would be at least at home for his treatment, but at his next visit he said that he had been increasingly able to do his work and that riding in an omnibus no longer gave him any pain.

Screening showed diminution of the bulge of the right lobe and some movement of the diaphragm.

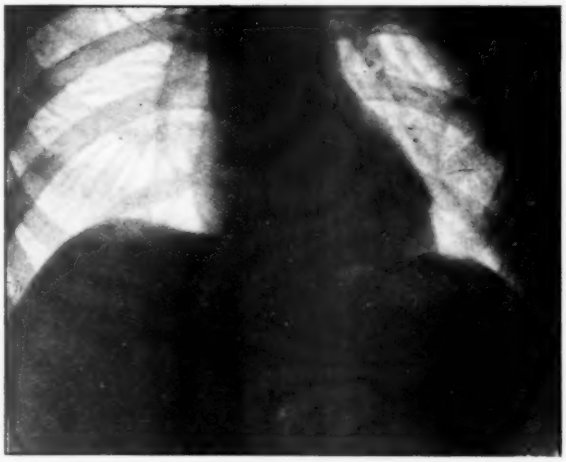


FIG. 2.

He was ordered to take emetine 1 gr. on April 8, 9, and 10, and again on April 15 and 16.

He was seen again on April 22. There was free movement of the diaphragm. Percussion was normal and breath sounds could be heard down to the eleventh rib behind.

The second picture was taken on April 22 and shows a very marked difference since the commencement of treatment (fig. 2).

His only complaint on April 22 was that he had some feeling of tightness over the liver.

His complexion, appetite, and general feeling of health were normal.

72 Parkes Weber: *Pituitary (Cystic ?) Tumour*

Pituitary (Cystic ?) Tumour.

By F. Parkes Weber, M.D.

The patient, W. E., a clerk, aged 47, has a pale, cachectic appearance with somewhat wizened, shrivelled face; moderate anæmia; more or less chronic headache (especially in the upper occipital region); bilateral temporal hemianopia; and (by Roentgen skiagrams) considerable antero-posterior enlargement of the pituitary fossa. The Wassermann reaction is negative. He has no axillary hair and only scanty hair on pubes, eyebrows and face; he never has had more, he says. Has had four children (all living and healthy). He has been sexually impotent for the last year. The testes are rather small. Brachial blood-pressure, systolic, 126 mm. Hg; diastolic, 88 mm. Hg.

He dates his present illness from an attack of influenza about February, 1925, which was followed by "weakness of eyesight." In the summer and autumn of 1925, when he came under observation, his symptoms were very similar to what they now are: anæmia, headache, enlargement of the pituitary fossa and bi-temporal hemianopia. Afterwards great improvement took place and in February, 1926, there was no definite hemianopia (Dr. C. Markus). I suggest that the temporary improvement may have been due to the leakage of a pituitary cyst into the cerebro-spinal fluid. The facial *gerodermia* in the present case I regard as a chronic pituitary symptom, analogous to the acute cachexia of Simmonds's "cachectic hypophyseal syndrome." Some of the cases of Rummo and Ferrari's type of "genito-dystrophic gerodermia" have been supposed to be due to congenital syphilis. In the present patient, though the Wassermann reaction is negative, there is the history of a doubtful syphilitic chancre in 1900.

Postscript (June, 1927).—The cerebro-spinal fluid gives a negative Wassermann reaction and shows nothing abnormal. There is no polyuria. Blood-sugar (May 17): 0·082 per cent. The blood-sugar curve is not abnormal.

In spite of the history regarding the deficiency of facial, pubic and axillary hair, I am inclined to regard the present chronic symptoms as due to a previous acute lesion of the anterior lobe of the pituitary gland, possibly connected with the "influenza" in 1925. The pituitary gland in the present case may have been affected as a *locus minoris resistentiæ*. Viewed in this light the case is an incomplete form of Simmonds's "cachectic hypophyseal syndrome," and is due to embolic or thrombotic necrosis of only a portion of the anterior pituitary lobe, resulting in cyst-formation (in the chronic stage of the condition). Probably some cases of the Lorrain type of infantilism ("ateleiosis" of Hastings Gilford) are due to similar lesions (possibly leading to cyst-formation) but arising in early life. The condition described by Hastings Gilford as "progeria" is perhaps due to a similar, *but more complete*, destruction of the anterior pituitary lobe in early life.—F. P. W.

Clinical Section 73

Abdominal Lymphogranulomatosis Maligna (Hodgkin's Disease), with Very High Blood-Eosinophilia.

By F. Parkes Weber, M.D.

The patient, H. W., aged 36, an English printer, complains of general weakness, and has a chronic remittent type of pyrexia (temperature usually up to 101° F. in the afternoons). There is no enlargement of superficial lymphatic glands, but in the abdomen, to the left of the umbilicus, is a deep, hard immovable swelling, probably retroperitoneal. The liver is not enlarged. The spleen cannot be felt, but is enlarged to percussion. Repeated blood-counts show slight anæmia and a blood-eosinophilia, averaging 40 to 50 per cent. of the total white cells. The Wassermann and Pirquet reactions are negative, and the intracutaneous reaction with an echinococcus antigen is likewise negative. The reaction for occult blood in the fæces is positive. Some muscular atrophy in the left upper extremity might be the result of left-sided infantile hemiplegia, of which, however, no history can be obtained. The patient thinks that the present illness commenced early in April, 1927, with pain in the left shoulder and general weakness. When admitted to hospital on April 23, he did not know that there was any difference in size of muscles between his two arms, but he complained of loss of power in the left hand and arm, which is now less marked. The pain in the region of the left shoulder has likewise passed off. The symptoms in the left upper limb may therefore have been really of recent onset and possibly of lymphogranulomatous origin. The deep reflexes in all four limbs are active, apparently slightly more active in the left than in the right arm.

The latest blood-count (May 10) shows somewhat less eosinophilia: Hæmoglobin, 66 per cent.; erythrocytes, 4,496,000 per c.mm. of blood; white cells, 8,300 (eosinophils, 26 per cent.; polymorphonuclear neutrophils, 59 per cent.; lymphocytes, 7 per cent.; monocytes, 8 per cent.).

Examination (including X-ray examination) of the thoracic organs shows nothing abnormal.

Professor W. Bulloch has kindly told me of an unpublished case of lymphogranulomatosis maligna, with similar high blood-eosinophilia, in which the eosinophilia was absent in the latter part of the illness, when the patient was losing ground. In that case the intestine was found to be affected, and in the present case there may be an intestinal lesion, as there is occult blood in the fæces.

Postscript (June 25, 1927).—The hard mass in the abdomen rapidly increased in size and the patient became weaker and extremely emaciated. He died on June 18. The necropsy and microscopic examination showed lymphogranulomatosis maligna (Hodgkin's granuloma) of the retroperitoneal lymphatic glands, forming a large conglomerate mass in the abdomen, almost as large as a man's head, adherent to the vertebral column. There were two ulcerated plaques of lymphogranulomatous infiltration in the jejunum. Examination of the vertebral canal showed that the

74 Parkes Weber: *Abdominal Lymphogranulomatosis Maligna*

epidural fat over the posterior (dorsal) surface of the dura mater in the thoracic (especially upper thoracic) region was diffusely infiltrated with similar lymphogranulomatous growth. The case will be published in detail elsewhere.[1]—F. P. W.

[1] In regard to lymphogranulomatous infiltration of the epidural fat in the vertebral canal, see F. P. Weber, *Quart. Journ. Med*, Oxford, 1923, xvii, pp. 1-5, and *International Clinics*, Philadelphia, 1926, series 36, i, pp. 127-136. The reason why in such cases sensory or motor symptoms may possibly pass off or be intermittent (independently of X-ray treatment) is probably to be sought in the observation that lymphogranulomatous growths may vary in size from time to time, doubtless owing to alteration in vascular turgescence. In connexion with the eosinophilia in the present case it should be added that during the last week or so of life there was intense universal pruritus with scratching.

Section of Dermatology.

President—Dr. J. H. SEQUEIRA.

CASES.

Lupus Erythematosus, treated by Krysolgan.

By H. C. SEMON, M.D.

PATIENT, female, aged 53. First seen January 11, 1927.

History. 1910.—First noticed spot on tip of right ear, and on end of nose.

1911.—Spread from ear to side of neck and across face from tip of nose to naso-labial groove. Condition persisted almost uninfluenced by various local applications.

1915.—Condition grew worse and carbon dioxide snow was used. New lesions appeared behind ears and on back of neck.

1919.—Admitted to hospital; tonsils removed. An autogenous vaccine was cautiously administered until April, 1920. No benefit was derived from the procedure.

No further specific treatment was attempted until she was admitted to the Royal Northern Hospital on January 13, 1927.

Condition on Admission.—Stout, pallid, and rather breathless: presystolic bruit at mitral area, and a few crepitations at bases of lungs.

She gave a history of scarlet fever in 1887, cholera in 1889, malaria in 1905, rheumatic fever in 1907, pleurisy in 1908-9, pneumonia in 1918.

A von Pirquet skin test resulted in a violent positive reaction, which was still in evidence *two months later*, and appears to prove a tuberculous diathesis.

Active lesions of lupus erythematosus on face, scalp, back of the neck, forearms, backs of hands, hypothenar eminences, thighs, and front of legs. They were of a relatively superficial type, consisting mainly of pink scaly macules, small in size and scattered irregularly, but in a rough symmetrical manner, over the areas indicated. There was a marked scarring of the nose, mainly due, it was believed, to the frequent applications of CO_2 snow, in 1915. Scars resulting from this cause are also apparent on the neck.

Treatment.—The patient was kept in bed, and the urine was examined daily for albumin, which is frequently present as a result of krysolgan injections. The injections were given intravenously in water—from 1 to 2 c.c.—at intervals varying from a week to fourteen days. No albumin was found at any time, and no ill-effects were observed except rheumatic pains, which were somewhat severe after the second and third injections. A simple zinc cream was the only local application used throughout the treatment. The krysolgan was injected for the first time on January 19, and the following is a tabulated account of the subsequent injections, their doses and the results obtained:—

January 19, 1927.—Krysolgan, 0·015 gr.

January 21, 1927.—Rheumatic pains in neck, shoulders and knees. No fever. No albuminuria.

January 24 1927.—Increase of itching (to which she is subject) in lesions on forearms. Commencing clearance of some of the patches on the neck.

Krysolgan.—0·15 gr.

January 26, 1927.—"Muzziness" in head; rheumatic pains clearing up.

January 31, 1927.—Krysolgan, 0·375 gr.

February 1, 1927.—Feels ill this morning. No pyrexia. Rheumatic pains in knees, feet, and left hand.

February 2, 1927.—Pains almost disappeared.

February 3, 1927.—Krysolgan, 0·75 gr.

February 15, 1927.—There are still rheumatic pains, but all lesions are now definitely clearing. *The effect of 0·6 gr. of thiosulphate of soda, injected intravenously, in 5 c.c. of water, was very striking.* Within two or three hours the pains had vanished and the patient slept undisturbed for the first time since the treatment was begun.

February 16, 1927.—The pains returned after a trial of the carbon-arc total bath, treatment with which was therefore abandoned.

February 23, 1927.—Krysolgan, 0·75 gr.

March 1, 1927.—Still inclined to rheumatic pains.

March 9, 1927.—Complete involution of all the lesions. The scarring is so slight as to be almost invisible on the forearms and back of the neck, and compares extremely well with that left by the old treatment with CO_2 snow.

The results in this case are comparable to those obtained in a somewhat similar case shown at the Meeting in December, 1926.[1]

In that case the patient's husband, himself a medical man, reported that there was now "but little trace of the old trouble (present for four years, and getting steadily worse) . . . in addition her general health has steadily improved . . . has more strength and energy . . . capable of sustained effort, etc.; it is as if the skin trouble had been a manifestation of a general infection, and that the injections had eliminated this as well as clearing the skin."

In this case there was also a definite history of tuberculosis, and it seems from a study of other cases, not yet reported, that krysolgan is likely to be of considerable therapeutic value in lupus erythematosus in which the factor of tuberculosis is associated.

Discussion.—Dr. H. W. BARBER asked what was the maximum dose of krysolgan that Dr. Semon had used. He, personally, had never given more than 0·375 gr.

Dr. SEMON (in reply) said that the largest dose of which he had had experience was 1·25 gr., and that had been followed by unwelcome symptoms; there had been much pain and collapse on the following day, and in consequence the patient had been kept in bed for two days. There had not been any rise in temperature. He had resolved not to exceed 0·75 gr. in future.

Case of von Recklinghausen's Disease without Tumour Formation.

By A. M. H. GRAY, C.B.E., M.D.

THE patient, a boy aged $8\frac{3}{4}$, has attended hospital under Dr. Cockayne's care since April, 1921. When he was 1 year and 9 months old his mother noticed that his neck was becoming pigmented so that she "could not make it clean." Small brown and black spots began to appear and the dark areas were spreading downwards to the trunk and arms. The boy has always had good health but is somewhat backward. No family history of pigmentation or tumours was obtained.

At the present time the patient shows:—

(1) Diffuse *café-au-lait* pigmentation, most marked round the neck, but also visible on the face and trunk.

(2) Irregular brownish patches, sharply differentiated from the surrounding skin, on the upper part of the chest and back, and showing a distinct linear distribution on the right arm.

[1] *Proceedings*, 1926, xx, 566.

(3) Scattered pigmentary spots, brown and black, mostly lentil-sized, on face, neck, trunk and limbs.

There is no pigmentation of the mucous membrane.

No evidence of tumour formation can be found nor are there any neuro-fibromata. No other signs are observable.

The case suggests von Recklinghausen's disease without tumour formation, of the type described in the report of a case by Dr. Parkes Weber (*Brit. Journ. Derm.*, 1909, xxi, p. 49), and also in a somewhat similar report by Wise (*Arch. of Derm. and Syph.*), in which three members of a family had this disease, but only one showed pigmentary changes.

In the present case the distribution of the pigmentation rules out xerodermia pigmentosa and Addison's disease. The latter condition is further ruled out by the duration of the pigmentation, the progressive development of which has been noted for practically six years.

Possibly tumours may appear at a later date.

Discussion.—Dr. F. PARKES WEBER said this case was almost exactly similar to one he first demonstrated in 1905, at the (old) Dermatological Society of London. Since that date the patient then shown had developed the typical complete form of von Recklinghausen's disease, not only with molluscous fibromata scattered about the body, but also with what seemed to be a plexiform neuroma on the right side of the neck. He believed the present case represented an early stage of slowly progressive von Recklinghausen's disease, not a permanently incomplete form. Several such cases besides his (Dr. Weber's) own case had been followed up over various periods of time.[1]

Dr. J. D. ROLLESTON said that in a series of cases of familial von Recklinghausen's disease which he had shown before the Clinical Section in 1911,[2] the father was what Dr. Parkes Weber described as a "full-blown" case, and some of the children showed the *café-au-lait* patches without tumours, while one child had the tumours and the *café-au-lait* patches; every stage of the disease had been present in that family.

Dr. A. WHITFIELD said that some years ago a series of cases of the disease were published which had been successfully treated by giving thyroid internally, but he himself had never found thyroid of any use in von Recklinghausen's disease. If given, however, at the stage before growths appeared it might possibly prove of value.

Dr. A. M. H. GRAY (in reply) said he would follow Dr. Whitfield's suggestion, and report the result.

Case of ? Angioma Serpiginosum: for Diagnosis.

By F. PARKES WEBER, M.D.

THE patient, A. E. F., aged 21½, English, employed in a clothing workshop, is a rather delicately-built young woman. Since May, 1926, small, discrete (scattered or grouped) cutaneous lesions have gradually appeared on the ulnar portion of the right upper extremity between the hand, near the wrist, and the middle of the upper arm. The individual lesions are red, reddish-brown and brown spots and minute patches, varying in shape and size, from two to twelve millimetres in their maximum cross-measurement. Most of them are aggregated in groups of two or three. Apparently when they first appear they are slightly elevated erythematous papules, like the smallest lesions now present on the ulnar portion of the back of the hand near the wrist; but they tend gradually to become brownish and slightly shiny and level with

[1] *See* F. Parkes Weber, "A Case of Recklinghausen's Disease, shown in 1905, as an Early Pigmentary *Forme Fruste*," *Proc. Roy. Soc. Med.*, 1926-1927, xx (Sect. Derm.), 22 (with illustration).

[2] Rolleston, J. D., and Macnaughton, N.S., *Proceedings*, 1911, iv (Clin. Sect.), 75, 114; *Rev. Neurol. and Psych.*, 1912, x, 1.

86 Weber: *Angioma Serpiginosum;* Drake: *Erythromelalgia?*

the surrounding skin, like the older lesions on the forearm and upper arm, apparently in process of involution. The largest cluster on the forearm was the first to appear (May, 1926). None have as yet completely disappeared. A few of the larger and browner lesions are dotted with red pin-point telangiectases. The patient's attention was first directed to them by actually seeing them; she is quite certain that there has never been any pain, itching, or abnormal sensation connected with them. The lesions have remained entirely confined to the right upper extremity.

The thyroid gland is somewhat large and there is very slight anæmia (4,000,000 erythrocytes to the cubic millimetre of blood), but otherwise, by examination of the thoracic and abdominal viscera, eyes, mouth and fauces, there is nothing special to note, excepting a trace of albumin in the urine. The brachial blood-pressure is: systolic, 140 mm. Hg (possible influence of excitement); diastolic, 75 mm. Hg. There is no enlargement of the liver, spleen or superficial lymphatic glands. The Pirquet cuti-reaction for tuberculosis and the Wassermann reaction are both negative. Factitious urticaria and decided dermographia cannot be elicited. Menstruation began at 14 years of age, and is regular. The patient has complained lately of occasional throbbing over the right eye, but otherwise she seems not to have suffered from ill-health. She has considerable insensibility of the fauces, but is apparently not hysterical, and there is nothing specially noteworthy in her past medical history or her family medical history.

That the cutaneous trouble is of artificial origin seems unlikely. The appearance of the more recent lesions (namely, the minute erythematous papules on the ulnar portion of the back of the hand near the wrist) speaks, to my mind, very much against the supposition that by some means the patient may have herself induced the lesions. It should here be noted that she is right-handed. A suggestion is that it may be a peculiar form of lichen planus. A more probable suggestion, which has been made by several who have seen her, is that the lesions represent an early stage of "angioma serpiginosum" (Crocker's name for the "infective angioma" of Hutchinson). There is, however, no characteristic "ringed" arrangement, such as is met with both in angioma serpiginosum and in "purpura annularis telangiectodes" (Majocchi). It is furthermore remarkable that the disease, in the present case, has appeared at several different parts of the same limb widely separated from each other.

Discussion.—Dr. MACLEOD said that the multiformity of the lesions in this case, and the fact that they were unlike those of any other skin disease he knew, inclined him to the suspicion that this was a case of artefact.

Dr. H. C. SEMON asked whether angioma serpiginosum followed injury. He had a patient with a tuberculous sinus on the front of the chest which had been treated with ultra-violet rays. Subsequently there had appeared characteristic angioma serpiginosum on the chest, which apparently had been started by the old injury. The patient had received no X-ray treatment.

Dr. PARKES WEBER (in reply) said that the presence of the small reddish papules on the hand, which were apparently the most recent lesions, was very much against the idea that the lesions had been artificially produced.

Case of (?) Erythromelalgia: for Diagnosis.

By J. A. DRAKE, M.D.

C. F., AGED 58, married, nine children, four living (three died in infancy).

Past History.—1923: Laparotomy, chronic pancreatitis found; cholecyst-enterostomy performed. 1925: Portion of gut resected for adhesions.

Present Condition.—Pain and swelling in left foot. Dusky bluish red area on anterior part of dorsum of foot just behind second, third and fourth toes involving

these toes and also great toe, mainly the dorsal surface but slightly on ventral aspect. Both legs mottled, but affected foot much the colder. No œdema, but the local circulation is sluggish. Condition more marked in the dependent position. Patient complains of pain in the region especially in the nail of the great toe. Cutaneous hyperæsthesia over affected part and pain on pressure. Behind the area, slight diminution of sensation to light touch; apart from this no evidence of altered sensation. Knee-jerks active. Pulsation of dorsalis pedis artery cannot be felt in left foot; pulsation of femoral arteries is equal on both sides.

Blood-pressure.—Systolic, 190; diastolic, 100.

Wassermann Reaction.—Negative.

Urine.—Trace of albumin. A few pus cells. Blood-sugar normal. Complexion muddy; skin of arms dry and rather ichthyotic; eyebrows thin in outer thirds. Appearance suggests deficiency of thyroid secretions.

Discussion.—Dr. PARKES WEBER said that those who saw many middle-aged male Hebrew patients were fairly familiar with a purplish, painful, condition of the foot, which might be called erythromelalgia. It was invariably connected with obstruction in the arterial supply to the foot owing to thrombo-angiitis obliterans. This patient seemed to have arterial obstruction of some kind, because no pulsation could be detected in her left dorsalis pedis artery, though there was quite good pulsation in the right one. The condition of this patient's great toe might therefore be ischæmic, though not due to thrombo-angiitis obliterans ("Leo Buerger's disease"), which chiefly attacked Hebrew males. But what was the cause of the angry-looking, reddish, firm, tender swelling on the dorsal surface of the left metatarsus, just proximal to the third or fourth toe? He (Dr. Weber) suggested that it was a neoplastic or chronic inflammatory growth of some kind.

Dr. DRAKE (in reply) said that the patient had been examined by a surgeon, and skiagrams of the foot had been taken, but no abnormality had been discovered. Apart from the vascular element, there must be a strong neuritic affection, as the pain was a typical nerve pain, and the extraordinary cutaneous hyperæsthesia could not be due merely to a vascular condition such as thrombo-angiitis. "Erythromelalgia" was a vague term, used many years before these days of more exact knowledge, and he was not sure what it included.

Mycosis Fungoides.

By H. W. BARBER, M.B., F.R.C.P.

A. B., MALE, aged 58.

Previous History.—Syphilis forty years ago.

Present Condition of skin began five or six months ago on the chin, and has gradually extended. There has been very severe itching. The eruption is, I think, typical of mycosis fungoides:—

Face.—Infiltrated scaly brownish-red patches: one on the forehead is ulcerated and crusted.

Neck.—Similar patches with marked infiltration.

The arms, thighs and legs are covered with infiltrated, figurate, scaly plaques, many showing central islets of healthy skin. There is some ulceration of the patches on the arm.

The patient is evidently addicted to alcohol. Examination of the heart suggests auricular fibrillation.

Benign Lymphogranuloma (Schaumann).

By H. W. Barber, M.B., F.R.C.P.

Mrs. M. H., aged 69, was sent to me by Dr. A. F. Conder, of Cheltenham.

Nothing significant in family history. Patient's general health has on the whole been very good, and she appears younger than her actual age.

About ten years ago, nodules began to develop round the ankles, and Dr. Conder informs me that long before she came under his care she had suffered from what was termed erythema nodosum, which had, with some slight intervals, been troublesome ever since, chiefly on feet and legs, and to some extent also on thighs, hands, wrists, and even lower abdomen.

About three years ago, Dr. Conder removed some "tubercular glands" from the submaxillary region, and, later, others were treated successfully with X-rays.

A year ago patient complained of pain down the right sciatic nerve, and Dr. Conder removed "a lump about the size of a golf-ball," which was reported by a pathologist to be a spindle-celled sarcoma. It was thought that the growth arose either from the sciatic sheath, or from the intermuscular septum. The swelling recurred about nine months later in the same situation and was again removed. Recurrence again took place and deep X-ray treatment was given by Dr. Curtis Webb.

I saw the patient, March 7, 1927; a glance at her hands and wrists suggested at once the diagnosis of benign lymphogranuloma, and further examination confirmed that diagnosis. The condition at present is as follows:—

Hands and Wrists.—On the backs of hands and wrists are smooth bluish swellings, and the proximal parts of the fingers are similarly affected, so that the fingers appear to taper to their tips. The remains of a few discrete nodules are palpable on the wrists, and one nodule is present on the flexor surface of the first phalanx of the middle finger of the right hand.

Forehead.—There is one bluish circumscribed nodule on the forehead.

Lower Extremities.—On the buttocks and thighs are a large number of bluish nodules of varying size, at the back of the right thigh are two large scars at the site of the above operations. The legs, below the knees, are swollen and œdematous, there being definite œdema behind the Achilles tendon. Scattered nodules are present and on the left leg are two markedly projecting ones over which the skin is reddened. There is also a general reticular cyanosis, resembling that seen in erythema ab igne, over the lowest parts of the legs.

I have not seen sections of the tumours that were removed, but I presume that they were also large sarcoid nodules.

There are no physical signs of disease in the chest or abdomen, and the spleen is not palpable. Dr. Conder reports that there has been a chronic infection of the urinary tract by the *Bacillus coli communis.*

Bones.—Wrists, hands, ankles and feet have been examined by X-rays (Mr. Redding). The appearances are normal, there being neither diffuse rarefaction, nor any of the characteristic clear areas.

Chest.—No signs of pulmonary deposits and the radiographic appearances are such as one might find in any normal person.

The von Pirquet reactions to both human and bovine tubercle, the Wassermann reaction and the complement-fixation test to tubercle are completely negative (Dr. Eyre). I have not yet received the report of the blood-count.

Discussion.—Dr. A. M. H. Gray said this case was very interesting because of the question of nomenclature. What were Dr. Barber's views as to the classification? Had he seen this case without having first spoken of it with Dr. Barber, he would have said it was an example of the hypodermic sarcoid of Darier, as almost all these lesions began in the subcutaneous fat. The majority of those seen definitely infiltrated the skin, and many had a

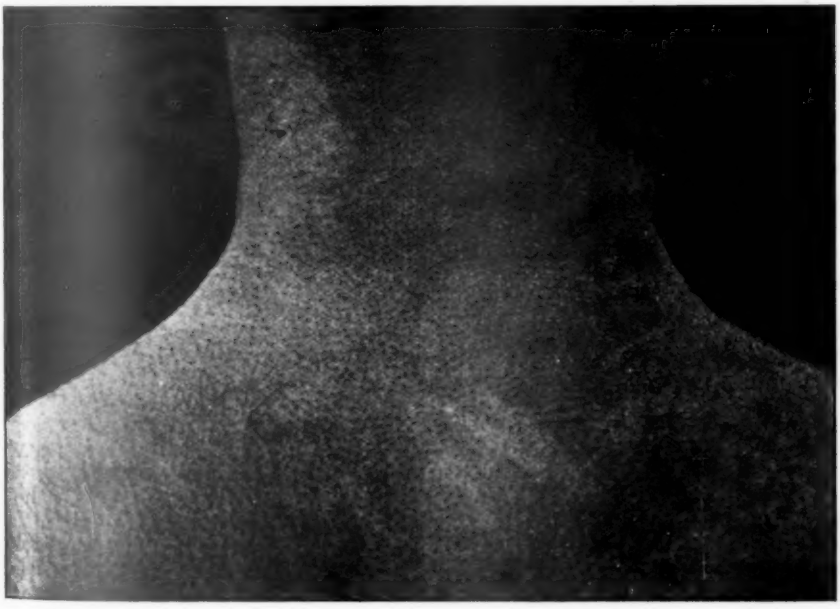
marked redness over them. None showed the curious transparency characteristic of cutaneous sarcoid of Boeck. It was still very uncertain whether these cases belonged to the group of benign lymphogranulomatosis of Schaumann, or to the true tuberculides. He considered many cases classified as Darier's hypodermic sarcoid to be simply examples of the subcutaneous type of lymphogranulomatosis benigna. The hands in the present case were suggestive of lupus pernio, but on feeling them one found little skin thickening compared with what was usually seen in this condition.

Dr. BARBER (in reply) said he was not quite sure what Darier meant by "hypodermic sarcoid." He (the speaker) admitted that some of the swellings in this case must be subcutaneous, for instance, those which were removed surgically. But on the buttocks were several which he would call dermic sarcoids; they seemed to be in the skin rather than in the subcutaneous tissue. Some time ago he had shown a typical case of lupus pernio with ordinary Boeck's sarcoid on the forehead (i.e., a bluish translucent swelling which was superficial), typical lupus pernio swellings of both cheeks, small lupoid nodules on the nose, and lupus pernio of the hands. There was extensive rarefaction of the bones, more extensive than in any other case he had seen. She (the patient) had deep nodules on the forearms and upper arms, exactly comparable to those seen in the present case. He would have said that the case under discussion was a potential one of lupus pernio. Schaumann said that subcutaneous sarcoid was a tuberculide, and that it had nothing to do with his benign lymphogranuloma. He did not think Schaumann was right in trying to draw a sharp line between some of these cases.

Keratosis Pilaris.

By J. T. INGRAM, M.D.

L. W., AGED 40, commercial traveller, was admitted to the London Hospital, under the care of Dr. Sequeira, in March, 1927, for a folliculitis of beard, axillary and pubic regions. For the past twelve months he had suffered from chronic



bronchitis, for which he received a course of thirteen injections of vaccine at weekly intervals from October, 1926.

During the course of this treatment he suffered from a generalized dermatitis accompanied by branny desquamation.

For six weeks preceding admission, the chest was rubbed with camphorated oil in order to relieve an exacerbation of the bronchitis. The patient, for one week only, had noticed a roughening of his skin, most marked upon the chest and gradually spreading to the rest of the body.

A slight, generalized ichthyosis is now seen, associated with follicular keratoses, most prominent upon the upper chest but present throughout the body, including the face and scalp. The palms and soles show hyperkeratosis with some desquamation. The nails are highly polished and show a ridge corresponding to the dermatitis associated with the vaccine therapy of October, 1926.

General examination of the patient shows some scarring with bronchiectasis of the left apex. There is no evidence of present tuberculosis.

The supervention of follicular keratoses in this case appeared definitely to have followed the inunctions with camphorated oil.

Sections from this case show generalized hyperkeratosis with plugging of the follicles, and chronic perifollicular inflammatory infiltration.

Grouped Comedones.

By J. T. Ingram, M.D.

Patient, H. O., a male infant aged 14 months, was seen in the Skin Department of the London Hospital by Dr. O'Donovan on March 1, 1927.

He then showed a group of follicular lesions, of two weeks' duration, covering the front of the chest.

The child had been in Bethnal Green Infirmary from August until the end of October, 1926, with meningitis and bronchitis. On discharge the mother thought there was some slight scaliness of the chest, of which she took no notice.

In February, 1927, the child developed a cough and the mother applied camphorated and eucalyptus oils and thermogene wool, intermittently, to the chest, during a period of two weeks. She then noticed the blackheads appearing, many of which subsequently became septic.

When seen at hospital the peripheral lesions on the chest were simple comedones, while the central lesions showed varying degrees of scabbed follicular pustules. No other members of the family were affected.

The case, I think, is one which readily falls into the class recognized as "grouped comedones in infants." Ætiologically it would appear that this case is a dermatitis venenata due to the external application of the oils mentioned—probably chiefly the camphorated oil—in a susceptible subject.

Camphorated oil, according to the British Pharmacopœia, should be made up with olive oil. Camphor may, however, be dissolved in mineral oils and does so appear on the market—though in this case the label should bear a note to this effect. Further, synthetic camphor, a hydrocarbon, may be used instead of the natural product.

I believe that the susceptible subject is the one with a tendency to follicular keratosis.

Crocker appears to have been the first to describe this group of cases, in 1884, though Colcott Fox showed one case before the Dermatological Society of London in 1883.

Most of the early cases recorded were those of grouped comedones upon the cheeks, forehead and scalp, dirt being regarded as the important factor, stress being specially laid by Crocker upon the dirty cap of the schoolboy. In his text-book, Crocker notes linseed poultices, and Sequeira camphorated oil as causative factors when the condition is seen on the chest. Ætiologically the condition cannot, apparently, be differentiated from acne of the adult due to the use of mineral oils.

Discussion.—Dr. A. M. H. GRAY asked whether it was suggested that camphor was essential to the production of the condition, or that impure oil had caused it. He had had the case of a child under his care in which grouped comedones had developed, and yet only pure olive oil had been used.

Dr. J. H. SEQUEIRA (President) said that grouped comedones had been seen after rubbing the skin with tallow.

Dr. INGRAM replied that in this case he suspected that the oil had produced the comedones.

Case for Diagnosis.

By HENRY MACCORMAC, C.B.E., M.D.

THE patient exhibited, a man aged 52, is by occupation both a farmer and an engineer; he resides in South Africa and has only recently come to England. With the exception of an attack of influenza in 1918 his previous health has been good. The complaint for which he now presents himself is considered by him to be of recent origin, but the atrophic changes in the skin of the scalp suggest a history of months rather than weeks. On the occipital region there are a number of bald, or semi-bald, areas, where the skin has been converted into scar tissue, the hair follicles being almost universally destroyed. Many comedo-like plugs are observed at the margin of these lesions, with, in addition, a few follicular spines. On the body, grouped over the shoulders and thighs, and to a less extent on the trunk, there are numerous follicular lesions, some presenting distinct spiny projections, others resembling comedones. The patient insists that the eruption on the body and limbs has developed during the last few weeks; it is accompanied by marked pruritus, which explains the presence of a number of secondary excoriations.

The exact classification of the eruption seems to present many difficulties. It has obvious affinities with folliculitis decalvans, with lichen planus, and with the cases shown by Dr. Graham Little under the name of lichen spinulosus.

Discussion.—Dr. G. B. DOWLING said the patient he showed at the Section was one of Dr. Barber's cases, and that Dr. Barber gave it the title lichen plano pilaris. There were quite characteristic lesions of lichen planus and lichen spinulosus. He did not see that patient until a year later, and then she came with cicatricial atrophy of the scalp. She still had such areas, which appeared to be permanent. He did not know whether, before atrophy of the scalp, she had follicular keratosis of that region.

Dr. J. H. SEQUEIRA (President) asked whether any Member had seen buccal lesions in these cases. (Dr. GRAHAM LITTLE: Not in my cases.)

Case for Diagnosis.

By WILFRID FOX, M.D.

THIS case seems to illustrate the group to which Dr. Whitfield has drawn attention, cases which appear to be those of pityriasis rosea developing lichen planus. The patient is aged 48, and she has had the rash six months. I queried her case being one of lichen planus when I first saw her fourteen days ago, on account of

92 Gray and Goldsmith: *Case for Diagnosis, ? Boeck's Sarcoid*

the long duration and absence of primitive plaque, but since then the scales have become less marked and the lichen papules more in evidence. The case is therefore in the transition stage. Syphilis has been excluded by the Wassermann proving negative.

Case for Diagnosis, ? Boeck's Sarcoid.

By A. M. H. Gray, C.B.E., M.D., and W. N. Goldsmith, B.Ch.

Dr. Goldsmith: This patient, a woman, aged 44, came under Dr. Gray's observation about four months ago, with the same appearance as you saw to day. She says that, since the lesions appeared over five years ago, there has been no material change in their appearance. There are several discoid infiltrated brownish-red patches on the forehead and temples; also one or two nodules along the margin of the right ala nasi. They are somewhat translucent in appearance, show a slight tendency to powdery scaling, are plateau-like and raised about $\frac{1}{12}$ in. above the skin surface. On diascopy yellowish staining remains but no distinct nodules. There is no tuberculosis in the family.

Wassermann reaction negative.

Tuberculin reactions (intradermic) + +.

Biopsy.—Portion of edge of one of the large plaques excised. Histological examination showed massive cellular deposits consisting almost exclusively of lymphocytes; only very few epithelioid and plasma cells were seen.

Blood-count.—Leukopenia (3,500 white blood-cells), lymphocytes 34 per cent., i.e., relative lymphocytosis, or diminution of polymorphonuclears.

Radiograms of fingers and toes showed no abnormality. Clinically, the lesions appear typical of Boeck's cutaneous sarcoid. Against this diagnosis are the positive tuberculin reactions and the nature of the cellular infiltration. The latter, together with the blood-count, point to a possible leukæmia. But the patient's general health is good; there is no general adenitis and the liver and spleen are not apparently enlarged. Lupus erythematosus can be excluded on the strength of the histology.

A short course of intramuscular injections of sodium morrhuate caused some malaise and nausea but had no effect on the lesions.

Dr. H. W. Barber said he had seen two such cases, and his experience was that the condition might undergo atrophy in the centre. Sometimes the lesions were like the superficial form of basal-celled carcinoma. One case had been seen by Dr. MacLeod, Dr. Adamson and himself, and Dr. MacLeod thought at first it was basal-celled carcinoma. He (the speaker) had a section made and sent to Darier, who said it was sarcoid. There was definite central atrophy, with scarring.

Section of Epidemiology and State Medicine.

President—Dr. S. Monckton Copeman, F.R.S.

The Influenzal Constitution.

By Sir William Hamer, M.D.

"As to the acute diseases (which I now purpose to discuss), some are generated by a secret and inexplicable alteration of the air infecting the bodies of mankind and are only conditioned by a peculiar crasis of the blood and humours in so far as the occult atmospheric influence shall have impressed the crasis upon the said bodies." ("Med. Obs." I, i, 6.)[1]

Certain sentences of Sydenham especially arrest attention:—

"The varied and inconsistent character of those diseases which we call epidemic." . . . "Their succeeding each other regularly and in series forming as it were a circle." . . . "One and the same disease in a year of one and the same constitution, exhibiting itself with various and dissimilar aspects as to its origin, its formation, its decline." . . . "Almost every constitution over and above the fevers it engenders having also a tendency to propagate with it some second disease from the class of proper and typical epidemics." . . . "Where we lose sight of our best guide Nature and give ourselves up to the luxury of guesswork we may have as many species of ailments as we choose to devise."

And last but not least the sentence quoted above.

We hear these sentences as students, but of course we have then our examinations to think about; we encounter them, perchance, as practitioners, but, being busy, are apt to pass by on the other side; there is a risk that as we grow older, thinking of Sydenham may merely serve to remind us of the novel use he made of Don Quixote, or of his dislike of the mountebank at Charing Cross, or of his lament concerning the old woman in every household, a practitioner, in an art she never learned, of the killing of mankind; we at least rejoice to know that the old Puritan rebel held his methods to be better than those of some other folks, as no doubt they were; and we cease to wonder at his being now and then querulous and impatient, for we recollect that he suffered much and lived in a trying age. At some time or other, however, we come right up against phenomena such as he described and we are then prompted to look again into the wonderful picture painted by him; the foreground assumes, now, relative unimportance; we are fascinated by the distant view, opening up glimpses into regions as yet unexplored; obstinate questionings arise within us and we become at any rate inspired with a saving consciousness of our own ignorance.

The events of the early nineties undoubtedly acted as a stimulus of this sort. Creighton's first volume appeared in 1891 and once more awakened interest in "the doctrine of an epidemic constitution and its counterpart doctrine of a predisposition in the human constitution" (I, 569). A sentence such as that at the head of this paper may be regarded as the first attempt at formulating the fundamental concept of immunology. Willis threw out a like suggestion in his chapter "On Epidemical Fevers" (xvii, par. 5). Both physicians, in fact, realized 250 years ago that there were infecting particles and a disposition of the blood and humours to react when acted upon by them. There was an interplay of conflicting forces and constitutions

[1] Dr. Greenwood's "reasonably strict translation" of Dr. Greenhill's Latin text of Sydenham is here quoted.

60 Hamer: *The Influenzal Constitution*

were thus engendered. Already in the eighties Sir William Collins insisted that "the germ was too much with us and the importance of soil absurdly underrated." Moreover, as Hirsch had testified, the English Health Department and the Epidemiological Society of London were redeeming the time. The two reports of Franklin Parsons and the writings of Bruce Low, McVail, Clemow, Bulstrode, Newman, and others, further demonstrated this. In 1893, Whitelegge's Milroy Lectures were delivered, while Thorne Thorne and Power, in the eighties, and Shirley Murphy in the nineties, steadily worked at "school influence," thus opening up the all-important question of "mass immunity."

At length, with the abundant data forthcoming from elementary schools, it appeared that a London measles curve could be constructed based on three elements: (i) an *incubation period* of fourteen days; (ii) an *infection factor* representing the number of persons infected on an average by each patient; and (iii) the *number of effective susceptibles*. [In measles the infecting power of the germ varies but little: (i) may be regarded as constant, and (ii) is determined by (iii).] To pass from the simple stable measles to the complex unstable influenza we must proceed by steps. Thus, a figment, as unreal as the dagger of the mind that led Macbeth on to his intent, may be conceived and implemented to construct preliminary scaffolding from which to build up an approximation to the true curve. The false influenza may (to simplify the problem) be assumed (during days, weeks, or months, according to nearness or distance from times of pandemic prevalence) to be *stable*, like measles, the only variable during the times in question being the number of "effective susceptibles." In pandemic prevalences of influenza in London the figures between which this variable ranges must be at least five times those (120,000-180,000) of measles; in influenza, moreover, protection is by no means lifelong, recurrence being frequent. Thus rate of addition of new susceptibles must also be increased perhaps in a similar proportion.

Whether these guesses are accurate or not makes little difference in estimating the duration of prevalences of influenza nearing the time of pandemic spread. Measles in London with its two-week incubation period has a wave-length of between eighteen months and two years; the corresponding elements in pandemic influenza may be taken roughly as one-third of a week (i.e., about one-sixth that in measles), and, say, $\frac{\text{two to four years}}{\text{six}}$[1] or one-third to two-thirds of a year. If the resulting curve be plotted out and compared with notifications in Copenhagen, it transpires that the imaginary curve differs notably from the real curve in two respects, (*a*) the rise is far more rapid in the real curve, and (*b*) the descent, though more tardy than the ascent, much steeper than in the theoretical curve.

Allowance must, therefore, be made not only for cyclically recurring modifications of resistance of the infected populations, but also for variations in the influenza germ itself, inasmuch as, unlike that of measles, it gains or else loses strength as it goes; at least it is clear that in the antepenultimate struggle towards the summit of each pandemic wave of influenza infective power is at its highest, the incubation period at its shortest, and the infection factor thus at a maximum; it is also fairly obvious that while, in the pandemic, well attested incubation periods of only a few hours occur and cases may prove infective even before the onset of symptoms, the heyday in the blood is much tamer in the precursors and trailers, tamest of all in the trough midway between the great waves. Apparently, indeed, in true influenza, making allowance for these variations in the germ and in its victims, we have to recognize:—

[1] A period longer than the corresponding period in measles must appear in the numerator, as the number of "effective susceptibles" in influenza is much greater than in measles, and in a large community the duration of the wave will of course be greater than in a small one.

(*a*) An average wave-length of one-third or two-thirds of a year at pandemic times.

(*b*) A somewhat longer wave-length, which owing to concomitant seasonal influences tends to be about a year, eighteen months or two years, for trailers and precursors nearly adjacent to the pandemic.

(*c*) A still longer wave-length which, when the average time from infection to infection becomes extended (as it apparently does, even to ten or fourteen days), may be estimated at some five to seven years in the trough between pandemics.

It is not suggested that these conclusions were accepted 250 years ago; still less that the fathers of epidemiology precognized Sir Ronald Ross's differential equations or the periodogram findings of Dr. Brownlee and his followers (or critics) Dr. Stallybrass and Mr. Spear; there can be no doubt, however, that study of epidemic waves has helped to bring us back again to Sydenham and sanity. All honour, therefore, is due to men who have climbed in this attenuated air of high mathematical altitudes, and in the present connexion especially to Dr. Brownlee, who has incidentally compelled acceptance by many minds of the view that influenza may be an endemic disease.

Our task is non-mathematical, just that of trying to straighten out some of the epidemiological difficulties. We know that the problem presented by the prevalences adjacent to the pandemics was in the seventeenth century readily solved by Sydenham and Willis (they were troubled by no avalanches), and that Creighton thirty years ago passed this obstacle by the device of linking up the agues and influenzas. We now see clearly, however, that on that steep slope of ice and snow leading to the North Col, which constitutes the only known practicable means of approach to our epidemiological Everest, there is a chimney blocked by a formidable crevasse, which may be taken to represent "influenza in mufti." Chalmers has pointed out that cerebro-spinal fever in an unrecognized form must lurk concealed in the intervals between the gross, open, palpable epidemic prevalences. Some have thought the "common cold" is the clue to passing the main obstacle itself; but it must be borne in mind that pandemic influenza affects not only the respiratory but also the central nervous and gastro-intestinal systems; the endemic influenzas are clearly likely then to be as catholic in their tastes as the pandemics, precursors and trailers; moreover, Creighton's linking of the influenzas with the "acute fevers" (agues) must be kept steadily in mind.

Before proceeding further it may be well to glance in retrospect at the slope, up which the pioneers have shown the way, and then (metaphorically speaking) to rope ourselves together, cut our steps and fix any necessary artificial aids which may help to make the difficult places epidemiologically secure. One lesson we can take to heart. Sydenham and Willis intuitively discerned truths which we find hard to grasp; but they saw their patients in a natural environment and studied the surroundings as well as the pathognomonic signs. The vastly improved appliances and technique of modern medicine, while facilitating clinical diagnosis, may distract attention from, and may indeed obscure, the epidemiological situation. Again, there is the modern habit of laying stress (in teaching) on the average or typical case. In the preface to the last edition of a well-known text-book it is stated that "the picture of disease which is described must be drawn from the average . . . frequently students pick up the exceptions in preference to the rules." In a systematic course on medicine which I was privileged to attend forty years ago, influenza was mentioned once only (the lecturer stating that pneumonia was "said to occur specially during an epidemic of influenza"); we were also told that typhus was practically an extinct disease. Beliefs regarding extinction of disease *must* be held conjointly with beliefs in the creation of new diseases, for Nature abhors a vacuum. So encephalitis lethargica was described as a new disease a few years ago, though no epidemiologist can possibly be under any illusions on that head.

Again, there are those who hold that association between epidemic diseases necessarily implies that they always prevail at the same moment of time, or failing this, at accurately spaced-out alternate times. Then, too, there is sometimes failure to recognize the fact that in Nature there is commonly a concurrence of favouring influences for the development of particular epidemics. On the other hand, as Hecker observes, "in any single isolated epidemic," Nature never "displays herself in all her bearings, nor brings into action at one time more than a few of the laws of general disease." In the case of influenza, as Crookshank says, "always in each apparently autonomous prevalence are all types represented, and for each type, in each prevalence, is there somewhere some one prevalence represented by predominance of that type."

When all this has been said we still wonder how it came about that Sydenham and Willis were so far ahead of their time. We cannot help sympathizing with Charles Lamb's friend, who felt that he could have written "Hamlet" if he had had the mind; but even if we had the minds of Sydenham and Willis, should we not still be considerably handicapped by the increased present-day complexity of interaction between germ and host?—owing, mainly, to more complete and widespread immunization, brought about by the crowding and greatly increased facilities of intercommunication of modern as contrasted with, say, seventeenth century life.

In fact, evolution of the germ itself has played a part in the drama of change from the homogeneous to the heterogeneous. The four English sweats were of fairly uniform type and were spaced out over long intervals (as influenza is nowadays in oceanic islands); in the latter half of the seventeenth century, in a rapidly growing London, there were on three occasions groups of differentiated prevalences within a few years; in cities in Italy, Germany, France and America, similar facts were noted; in the eighteenth century in places like Yorkshire, Plymouth, Edinburgh, Ireland, on the Continent, and in the United States, annalists described these epidemic constitutions, and the Bills show they existed in London, though the tangle there was becoming increasingly difficult to unravel; at the close of the eighteenth and in the early nineteenth century confusion was worse confounded and, here in London, the annals of continued fever became much more extensive and informative than those of influenza; after 1847, influenza in London was almost entirely missed for many years; and it devolved upon Goodhart to substantiate the claim of the "trailers" of the "nineties" to be accepted as influenzas. Those prevalences and the more recent ones again served to focus attention upon the influenzal constitution; indeed, it is agreed by many, nowadays, that in big centres of population the fire of endemic influenza is ever smouldering, in the form of a more or less chronic illness, mainly affecting "influenza subjects," and differing so greatly from the influenza of the pandemics that those who are so fortunate as to have no personal experience of it regard its relationship to the great influenzas with complete scepticism.

Dr. Greenwood summed up the situation seven years ago, saying, "The constructive work of the immediate future must be to render precise the still vague notion of an epidemic constitution." Before that he had urged that, to such an end as this, we should follow

> "the vicissitudes of some definite disease through a considerable period of time in order to realize the existence of secular factors; and then separately investigate the immediate features of various epidemics."

Sydenham had a similar idea in mind when he decided to begin with fevers. "Two-thirds of our race die of fevers," he said, and yet fevers "have no name founded upon the general character of the constitution," but are merely known by "changes impressed upon the blood or else from some symptom," . . . "I shall take as a starting point continued fever; I look upon this as the leader of the band."

Creighton, however, comments, "Such a work has not been made easier in all respects by the exact discrimination and perfected diagnosis to which we are accustomed in present day fevers. . . ." Between 1840 and 1850 "There arose the presumption that there were, and always had been, three forms of continued fever in Britain (spotted typhus, enteric and relapsing fevers) and that these might have been left on record by the physicians of former generations, if they had used the modern exactness and minuteness in observing both clinical history and anatomical state which were seen at their best in Sir William Jenner." To settle "the identity or non-identity of these diseases by reference to old writers" was, Jenner held, absurd (p. 90). And yet the modern view, as expressed by Dr. Greenwood, is that "the study of an epidemic, without reference to the previous history of the disease involved, can but lead to imperfect apprehension of the facts."

Jenner asked (p. 453) "If these diseases be now and have been for centuries different in symptoms, how came it to pass that they were so frequently associated together?" He says *first*, "symptoms of one are occasionally present in others . . ." *second*, "varieties of one class may simulate those of another class . . ." and *third*, "certain other diseases have not infrequently been associated with them." There is only one reference to influenza (a mere *obiter dictum*) in Sir William Jenner's book (p. 440); he tells us, however, that he suffered from typhus in 1847—apparently during the great influenza prevalence, and this fact may be significant of much. He clearly recognized the limitations of the "mulberry rash" as a guide to diagnosis (pp. 21, 22, 90, 413, 450). He pledged himself, however, "not to divide where Nature has drawn no line, nor to generalize where Nature has bestowed no unity"—but he studied merely the *clinical* side of the shield, neglecting its *epidemiological* aspect, which as Creighton tells us compels recognition of "gradations, modifications, affinities, being careless of symmetry, of definitions or clear cut nosological ideas, or the dividing lines of a classification." But worse remained behind; there was soon reached the situation described by Hueppe.

". . . The notion that every specific disease is caused by a specifically constant parasite is in accordance with the ontological wish of many physicians, who are thus spared the trouble of further reflection. . . ."

He considers, however, "that ultimately the variable as well as the constant will find its place, the mysticism of ontology having been taken away from both."

Recent happenings have facilitated this. The sun breaks through the clouds with Crookshank's volume of 1922 and the Ministry of Health Report of 1920, both of which truly mark advance. The Report says:—

"An epidemic constitution develops favourable to influenza" and there are concurrences, similarities and inter-relationships between outbreaks of cerebro-spinal fever, poliomyelitis and outbreaks of influenza, bronchitis and pneumonia" . . . "identifications of influenza must be based upon demographic or epidemiological grounds" . . . "there is no option but to establish a unity in diversity, to make the very diversity of its clinical forms a mark of influenza" (p. 4).

Accepting these guiding principles as our interpretation of the present, we may (so Professor Whitehead tells us) endeavour to interpret the past on that basis, and perchance incidentally throw some light upon "influenza in mufti" by following back the history of the conception "influenzal constitution." (See fig. 1.)

1915-26.—The "setting" of the 1918-19 influenzas was in the main one of pneumonia and bronchitis; diseases of the central nervous and gastro-intestinal systems assumed prominence in the precursors and trailers; in eastern Europe corresponding prevalences were usually described as "typhus," "relapsing fever,"

and "ague." The Great War may help to explain some of the peculiarities of this constitution. Special attention has been called by Stevenson, Brownlee and others to the unusual age-incidence observed in October-November, 1918. Brownlee, *Proceedings R.S.M.* (Sect. Epid.), 1925, p. 84, contrasts it with that in the large

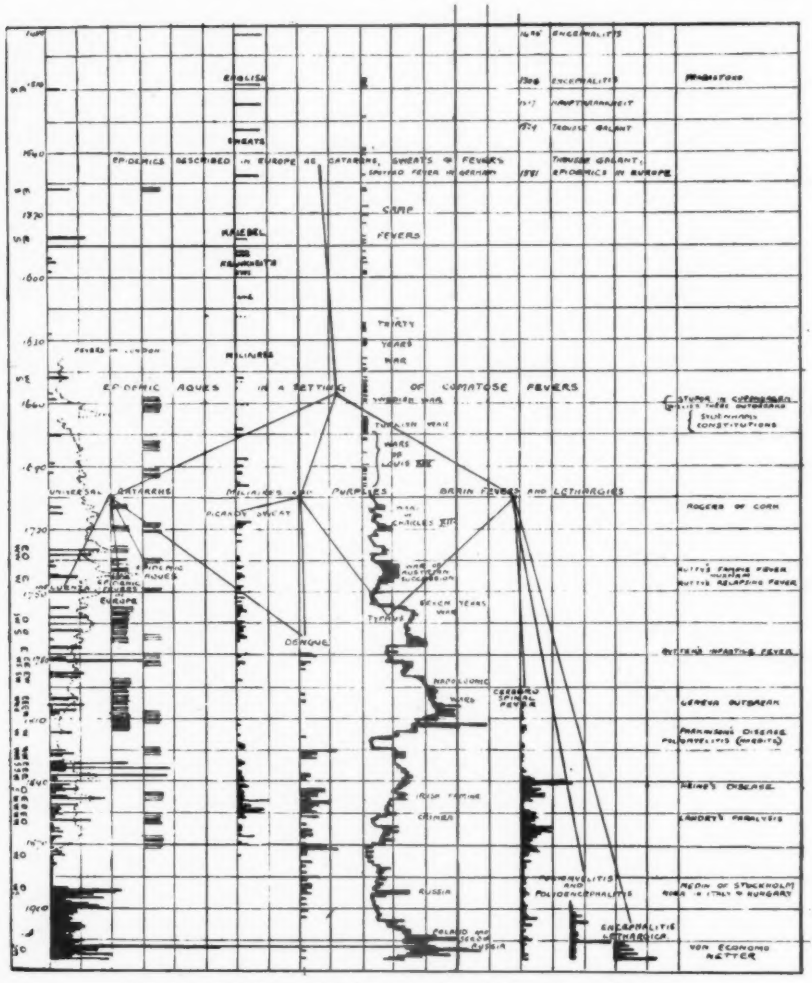


FIG. 1.—Evolutionary development of the influenzal constitutiion.

epidemics of the eighteenth century. A differentiating factor here may have been the degree of concentration of persons at "business ages"; under modern conditions of transport a considerable percentage of the population of Greater London, at those

ages, has in effect been added to that of London. The interval of time since a preceding pandemic also exercises influence in this connexion.

1889-95.—This "setting" has been fully illustrated by Crookshank and others. In north-west Europe the severe winters of the early nineties were associated with specially fatal pulmonary prevalences. Creighton (II, 397), says:—

"History does not appear to supply a parallel case to the four successive influenzas, in the period 1889-94, unless we count the seasonal epidemic agues of former 'constitutions' as equivalent to influenzas for the purpose of working out a series."

The relation of dengue to influenza, here, and in the 1915-26 "setting," is noteworthy.

1874-75 and 1858-59.—These pandemics were overlooked in this country, partly, doubtless, owing to "mass immunization" following the prolonged succession of influenzas next to be considered.

1831-47.—A time of rapid growth of population in London—with some privation in the early years—later the Irish famine overshadowed everything else. There were great influenzas in 1831, 1833, 1837 and 1847; preceded by a widespread "influenza" in America in 1826, by "relapsing fever" here in 1826-27, and by "acrodynie" in Paris in 1827-28; followed by much "typhus," and later (1865-66) by "cerebro-spinal fever." "Typhus" throughout was the dominant feature in the "setting" of these influenzas. In 1833 the more serious cases (Hingeston) "were marked by deep nervous depression." In 1836-37 the disease was apt to assume "a typhoid character," and mention is made (Streeton) of "meningitis, coma, affection of speech and delirium," while "inflammation of the spinal marrow" (Appleton) also occurred. Farr comments (10th Rep., xxxiii), on the association of influenza and typhus in 1847 and in previous epidemics. Graves remarks upon the facility with which "a simple cold (which in England would be perfectly devoid of danger) runs into maculated fever in Ireland," and says (p. 489): "many of those who died in the fever sheds were doubtless cases of influenza." He observes (p. 127): "I myself have never seen petechial fever epidemic in Ireland." Peacock's account of the 1847 influenza, says Creighton, reads like "an old constitution by Sydenham or Huxham . . ." he cannot refrain from acknowledging "that these several affections are not merely coetaneous but correlative, and types and modifications of one disease with which they have a common origin."

1815-16.—Influenza in U.S.A. "Relapsing fever," in London.

1799-1803.—A "setting" of "fevers" (see Creighton II, 159, 258, 373), "chiefly of the type designated typhus . . . (originating) from epidemic catarrh." Haeser speaks of their "typhus-artigen Charakter" in England. Symes Thompson's summary notes that "It seemed to participate of typhus" (198). "A typhus state" was "brought on after the tenth day." "At Hull, seemingly distinct disorders were included under the same name (influenza) from a firm conviction of their being different types . . . occasioned by the same cause." At Gosport and St. Neot's influenza "took on the appearance of typhus" in the workhouses. It spread from the Assizes at Shrewsbury and Exeter. In London "it degenerates into the typhus kind," and also at Bristol, in some cases "the attack could hardly be distinguished from that of ordinary typhus." At Bath it was complicated by typhus in "labourers, artisans and poor people who lived on bad food." In the Isle of Man it was so like typhus that "we were difficulted in the diagnosis."

"The Epidemic Agues of 1780-85."—So Creighton describes the "setting" of the influenza of 1782, which came at a time when the London Bills were entering upon their period of decadence. "Agues" held the field, though Carmichael Smyth says:

"the late influenza might very properly have been named the sweating sickness . . . relapses were common and the disease was apt to put on 'a putrid type'."

The College of Physicians found that:

"the distemper was by no means so uniform as to present the same identical appearances . . . nor yet so various but that the resemblance could be easily discovered."

This is an improvement on those latter-day ghostly manifestations, the "clinical entities" against which Sir Clifford Allbutt insistently waged war, and represents, indeed, some sort of approximation to the doctrine of Sydenham and Willis; but the accounts both here and on the Continent indicate considerable perplexity. Haeser (p. 527) says that some thought the influenza of 1782 brought a change of constitution; he deprecates, however, the growing use of the term "Nervenfieber" and holds that confusion resulted from the adoption of arbitrary hypotheses (he refers particularly to the wave of "Brownianismus" which swept over Italy and Germany). He inclines on the whole to suspect that there was some real change from the abdominal to the exanthematous "typhus-form." The age was one of revolution and of social and industrial changes. It is quite clear that there was notable improvement in the public health in London, and a decline in "fevers," but both here and abroad it was a dark and sterile period in epidemiology and medical statistics, though surgery and midwifery made notable advances; moreover, at the close of the century a commencement in hospital and dispensary provision was made and vaccination was introduced to the world by Edward Jenner.

1729-76.—This, like the middle third of the nineteenth century, was a period of growth in London, with occasional privation and much increase of intercommunication and traffic. Hamilton says it was "an era of luxury and enervation, of tea and gin; carriages are more frequent." "Typhus" disappears altogether in 1760, and "influenza" in 1743, but with the help of the annalists, and the Bills, it is possible to feel our way. The Ministry's Report of 1920 finds "the influenza periods of 1675-88, of 1729, of 1734 and 1743" set in "circumstances which are harmonious with one another," and with those of later periods. Creighton's Tables (based on the Bills) show (see fig 2) influenza rises on or near the summits (that of 1743 on the slope just beyond the summit) of longer waves of "fever"; the influenzas of 1762 and 1767 stand high on a great multi-annual "fever" wave; that of 1775 is on the declining slope of another, and Grant says "it was attended by the same comatose fever" which Sydenham associated with the epidemic catarrh of a century earlier. One of Farr's Tables (Vital Statistics p. 304) shows "fevers" declining and "inflammation" rising; the annalists describe "symptom complexes," which mimic the modern pulmonary, nervous and gastro-intestinal prevalences of influenza. Creighton has laid stress on the gradual development of fever dens in the great towns, and supplies instances "of country-bred people plunging abruptly" into them; of the periodic overflow of "volatile typhus" from them to surrounding populations; and of their activity as "forcing houses," a rôle which Simon allotted to camps, gaols and ships at a still earlier period. The worst kind of "typhus," says Creighton, affects "country people who crowded to the towns."

In the "sixties," and generally speaking throughout the middle third of the eighteenth century, "putrid and nervous fevers" prevail, there is mention of lethargy, stupor (Manningham, Wintringham, Hillary, Huxham), in France of "affections soporeuses," of relapses (Rutty, Gilchrist), of petechiæ and miliary rashes (Hamilton, Fothergill, Johnstone) and of pneumonia. Like phenomena are described in northern France by Lepecq, Bellot and at a later date by Rayer. All these writers seem to have been impressed with "the moving continuity of things." Epidemiologists from the time of Sydenham onwards have had to face conclusions to which a widely extended application is now given. As Bergson says, "We can only proceed according to the cinematographical method . . . What is *real* is the continual change of form: form is only a snapshot view of a transition."

The experts of to-day, writing on encephalitis, meningitis, etc., are generally prepared to throw their net quite widely and may go so far as to admit relationships between epidemic diseases of the central nervous system and the great catarrhal

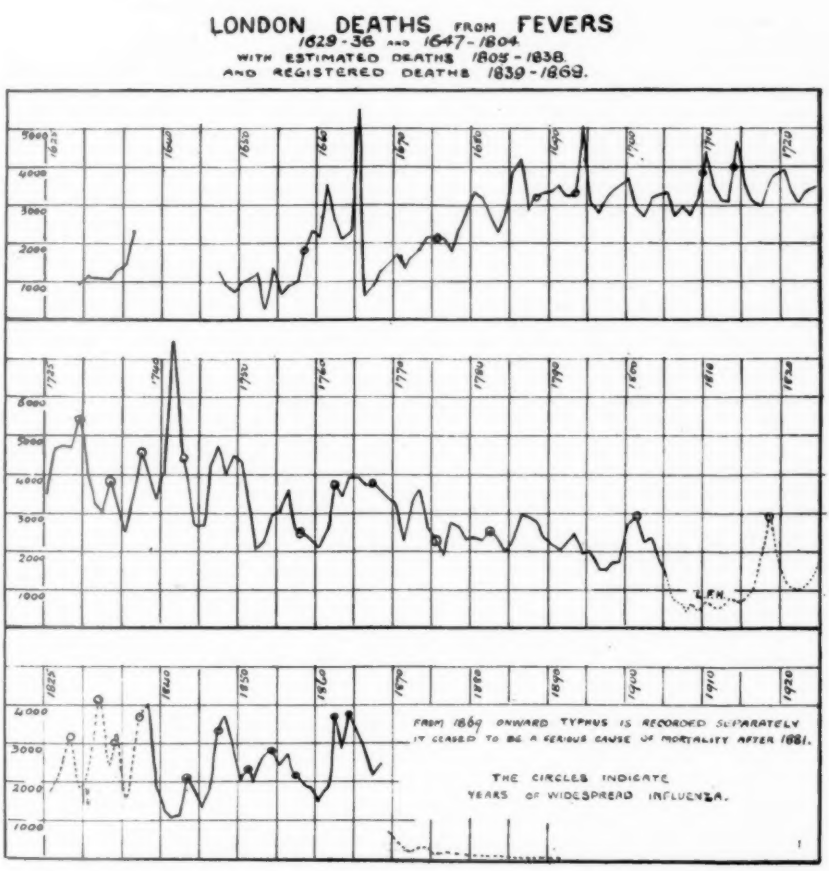


FIG. 2.—Setting of influenza on the crests of waves in fevers as seen in the London "Bills."

epidemics; but they are apt to be distrustful of epidemiological records, even of those of the early eighteenth century. Their feeling is, seemingly, that of

"the man lost in a thorny wood, who rends the thorns and is rent by the thorns, not knowing how to find the open air but toiling desperately to find it out."

They do not care to face "brain fevers," "soporosités," "comatose fevers," and "fièvres pétéchiales," and fear to press through the jungle of ship, camp, harvest and gaol fevers of two hundred and more years ago and penetrate into the primeval

68 Hamer: *The Influenzal Constitution*

forest of "miliaires," "fièvres pourprées," "fevers infestous to the brain and nervous stock," "trousse-galants" and "sweats." And yet, accepting the principle of continuity, and making allowances for changing terminology, we clearly discern "settings" of influenza corresponding to those of to-day, and find the notion that these prevalences make up constitutions firmly held by the epidemiologists of those times.

It may be useful, however, at this point to go right back to the fifteenth and sixteenth centuries, before the influence of Sydenham and his contemporaries was felt, and try to make our way thence to the middle of the eighteenth century, where the two tracks "influenza" and "typhus" first become recognizable and thereafter proceed in close companionship for the next hundred years.

At the outset we are struck by the resemblance of the complex of sweats, encephalitis, hauptwehe, trousse-galant, etc., round about 1485, to the modern influenzas. There is notably more concentration into pandemics (variously named), less spreading out into "trailers." In the Continental wars of the seventeenth and eighteenth centuries there was close association of epidemic agues and war fevers; Haeser notes that "Lagerseuchen" occurred "in der engsten Verbindung mit den Malaria-Fiebern." Soldiers of fortune, camp followers, harvest workers were implicated, just as the recruits in French barracks were later attacked by cerebro-spinal fever, and as, later still, Crystal Palace and camp outbreaks occurred; compare, again, the P.U.O.'s and new diseases of the trenches, and the transport catastrophes of American troops rushed across the ocean in the War. In the early centuries crowding and bringing together of strangers find special expression in gaol-fevers and ship-fevers. "Typhus," say some; a "volatile typhus" truly. The early "typhus" was, as a rule, "volatile," and it specially affected the "noblesse," "persons of the Court," "gens aisés," etc. Creighton says, "Influenza enters undoubtedly into the Protean infections of the sixteenth century, and is itself no small part of the Proteus." Already, in the "gentle correction" of 1580-2, which was associated with "hot agues," there were lasks, lienteries and dysenteries anticipating modern gastro-intestinal influenzas. We see, as in a glass darkly, our modern pandemic phase, with associated stupors and epidemic pneumonias, "featuring" cerebro-spinal and pulmonary trailers; but more closely than all these, their lineal descendants the early prevalences resemble their predecessor the English sweat.

Creighton describes "new diseases" in 1612-13, 1623-24, 1625, 1638-9, 1643-4 and 1651,

> "not one of which was an influenza as we understand the term, but they were certainly as mysterious as any epidemics admitted in the canon of influenza."

He writes (i, 409):—

> "Influenza is the only sickness familiar to ourselves which shows the volatile character, and we are apt to conclude that no other type of fever ever had that character. . . ."

But in the eighteenth century "spotted fever" rests "like an atmosphere of infection over whole tracts of Great Britain and Ireland," and "if we give the name of influenza to the epidemical 'hot agues' of the sixteenth century" we may "regard them as embracing (these) types of fever."

This brings us to Willis's "Fever and Influenza, 1657-59,"

> "of peculiar interest for the reason, that it is the first systematic piece of epidemiology written in England, and that the middle epidemic of the three was one of influenza." (Creighton).

The first prevalence was a brain fever, like those of 1915 and early 1918. The second came "as if sent by some blast from the stars" . . . "and laid hold of many

together." The third resembled the first. "In some on the first or second day came 'little broad and red spots'—the patients lay without speaking" (as in Sydenham's "absolute aphonia" of 1673-6 or Butter's "acute fever with dumbness" of 1782). "The exanthem," says Creighton, "reminds one more of the rash of sweating sickness or dengue than the spots of typhus." He adds,

"The strangest part of these narratives is not the catarrhal influenza . . . but the prevalence of anomalous fevers . . . in respect of contagion, spots, pains and other symptoms, like typhus. There are many more experiences of the like kind in the years to follow."

Willis's fever of 1661, "chiefly infestous to the brain and nervous stock," links the 1657-59 prevalences with Sydenham's first constitution, 1661-4, which was identical with his later constitution of 1678-80. Two full cycles, in fact, are completed by adding Willis's prevalences to Sydenham's (including the outbreaks referred to in the Brady letter) and to those described by Molyneux and Morley in 1688 and 1693.

Creighton, carrying the record forward, observes

"For Wintringham, Hillary, Huxham, Rutty and Warren, as for Willis, Whitmore and Sydenham, nearly a century earlier, it was all one aguish constitution."

At length the name influenza was formally adopted by the College of Physicians and this put an end "to the ambiguity between agues and influenzas, leaving the curious correspondence between them in time and place, or the nosological affinities between them as interesting as ever" (II, 362).

We have now traced the evolution of that "moving continuity," the influenzal constitution, from the English sweats to the multi-annual cycle of change of the London Bills, and have seen how the brain fevers, etc., which make up these early "settings" of influenza, gradually merge into the typhus, relapsing fever, pneumonia, etc., of 100 years ago; while, still later, the two first become supplanted, in their turn, by certain epidemic diseases of the central nervous system. The evolutionary process may, perhaps, be best illustrated by focusing attention upon a single populous area, such as London. It may be described in rough outline thus:—

Century		Population	Season of year	Setting of the Influenzas	Clinical types of prevalence
15th and 16th	English sweats	About 50,000 to 60,000	Always in summer	Isolated prevalences, the infection dying out for periods of ten or more years	A composite portrait of the three as yet not completely differentiated modern clinical types.
17th	Sydenham's constitutions	About 400,000	Sometimes in winter	A constitution of three or four prevalences	Pulmonary and cerebrospinal types are now distinguishable.
18th	1729-1776	About 600,000	Usually in winter	Constitution embraces a period of five or six years	A catarrhal widespread prevalence appears at or near the crest of waves of fevers. The "typhus" state is more particularly met with under conditions of privation and overcrowding. On the Continent—raphanias, convulsive ergotisms, miliaires, and sweats, appear in substitution for the "fevers" of London.

70 Hamer: *The Influenzal Constitution*

Century		Population	Season of year	Setting of the Influenzas	Clinical types of prevalence
19th	1831-47	3,000,000	Almost always in winter	Length of time covered by associated prevalences further extended	Connexion of "fevers" with "influenza" clearly recognized by Farr, Graves, and others. The three types, moreover, are now explicitly described.
	1890-95	4,300,000	Do.	Do. do.	The three modern types (pulmonary, cerebrospinal, and gastrointestinal) of associated prevalence, generally recognized by epidemiologists.
20th	1915-27	4,500,000	Do.	Do. do.	The pulmonary type of associated prevalence almost universally recognized. In Eastern Europe, "fevers" (typhus and relapsing) also described. In the rest of the world, as in London, their place is taken by epidemic diseases of the central nervous and gastrointestinal systems. Endemic influenza now becoming recognized.

London's experience shows that the season of the year at which the prevalence occurs has much influence (the old summer incidence was doubtless largely determined by the fact that summer was the time when free intercommunication between the Continent and this country existed); further, rapidity of growth of population in a given area is of great importance, as helping to determine the extent to which endemicity of the disease is favoured; over and above this, however, there is a gradual emergence of differentiated types of prevalence, as the centuries pass. It will be noted that in the seventeenth century there were associated outbreaks of a type not yet markedly differentiated from the central influenza; hence the comparative simplicity of the problem then, as compared with the present day, with its much more differentiated precursors and trailers, to say nothing of the still more elusive endemic influenzas.

It is now necessary carefully to look into the validity of some obvious criticisms regarding the conceptions outlined above. The relapsing fever of to-day has been held (since 1873) to be associated with a spirillum, and "typhus fever" is sometimes diagnosed on the strength of the Weil-Felix reaction, while "typhus blood" is found to produce a rise of temperature in monkeys. The question thus arises, how to explain the fact that in the last twelve years in Poland, Serbia, and Russia the "setting" of influenza has been in marked contrast with that obtaining elsewhere, inasmuch as in the three countries named typhus and relapsing fever have reappeared in their original rôles. As regards Russia, as Dr. W. H. Gantt admits, very little is known and practically nothing about its "influenza"; but many observers, among them Members of this Section, have studied the typhus and relapsing fever of Serbia and Poland.

There is surely need here for recognizing the "correlativity" of certain (symptom-complex) "*opposites*," and realizing that they are "inseparable elements of a higher

(epidemiological) *unity*." The case for such a "unity of opposites" may be based upon three grounds.

First.—There are the individual epidemiological experiences which strongly influence the minds of those who may chance to encounter them. "Seeing is believing!" The cerebro-spinal fever and poliomyelitis outbreaks of 1915 and onwards, studied by Reece, Farrar and Dorgan, and in London Annual Reports, and the lectures and essays of Crookshank may be cited. Another case in point is the following. At the time when famine and war were attended by great epidemics in Eastern Europe, the inmates of the London mental hospitals, suffering from food and coal shortage *plus* special epidemic influences, showed very high mortality, attributed in the main to influenza, pneumonia, dysentery and typhoid fever. Questions were raised straightway as to the deaths returned under the two last-named headings (London Annual Report, 1920), and it was pointed out that a like association of dysentery and typhoid fever had been commented upon in 1899-1901 by Mott and Durham in London mental hospitals, and by the Commissioners in the South African War. The curious point is that there was no more thought of associating these "fevers," in London and South Africa (in 1899-1901) and again in London (1915-20), with the "influenzal constitutions" of those years, than there was (at the later date) in connexion with the typhus, etc., of Poland, Serbia and Russia.

Second.—Throughout the period of statistics the London curve shows the influenzas forming as it were the spray on the crest of each wave of "fevers." This has already been shown to hold good for the Bills in the eighteenth century. It can be seen, too, though less clearly, in death-rates since 1841 (see fig. 3) The contour for "all causes" is of course very irregular (notably in years of cholera and smallpox epidemics, in less degree in those of "typhus" and of epidemic throat diseases). If a contour for "all causes *less* zymotics" be plotted out, the undulations of the eighteenth and early part of the nineteenth century are apparent, although less strikingly than before 1841, for some "fevers" have no doubt gone with the deleted zymotics. These undulations are not entirely lost, though they become of course less marked, after abstracting in addition "pneumonia and bronchitis." The black circles on the summits of the fever waves represent influenzas (1847, 1851, 1855, 1858, 1862, 1864, 1875, 1890-95, 1899, 1908, 1915, 1918). Thus influenza always has its "setting," whether it be of "fevers" (as in London from 1700-1847 and in Eastern Europe in recent years) or of pneumonia, bronchitis, and epidemic diseases of the central nervous system (as in most civilized lands during the last half century).

Third.—"Typhus" cannot be regarded (speaking epidemiologically) as a clinical or pathological entity, distinguishable by a pathognomonic sign, mulberry rash, Weil-Felix reaction, or result of monkey inoculations. The "monkey-typhus" is a new universe of discourse, and it is not legitimate to apply its findings to the old universe. The chief "typhus" areas of inoculators of typhus blood have been Algeria and Mexico, and, with Murchison, the "typhus" of those countries was gravely suspect, none of the descriptions coming therefrom satisfying him as to the nature of the disease. Fletcher and Lesslar have recently described a "tropical typhus," which can be diagnosed by employment of the Weil-Felix reaction, but they tell us that its "epidemiological features are fundamentally different" from those "of the great epidemic disease." Miss Robertson judicially says, "In the case of the monkey I am myself convinced there is no doubt whatever that the injection of virulent typhus blood produces a marked and clearly defined febrile reaction." Sir William Jenner might, however, have expected a demonstration of his pathognomonic rash. Murchison (pp. 131-2) discusses "petechiæ," a term which, he says, "is used in very different significations and hence has arisen great confusion." The

72 Hamer: *The Influenzal Constitution*

Irish and Continental physicians were, as a rule, of opinion that the special rash could not be regarded as peculiar to typhus fever. Corfield notes de Claubry's view, that in typhus and typhoid " the lenticular eruption " may be " the same," and adds

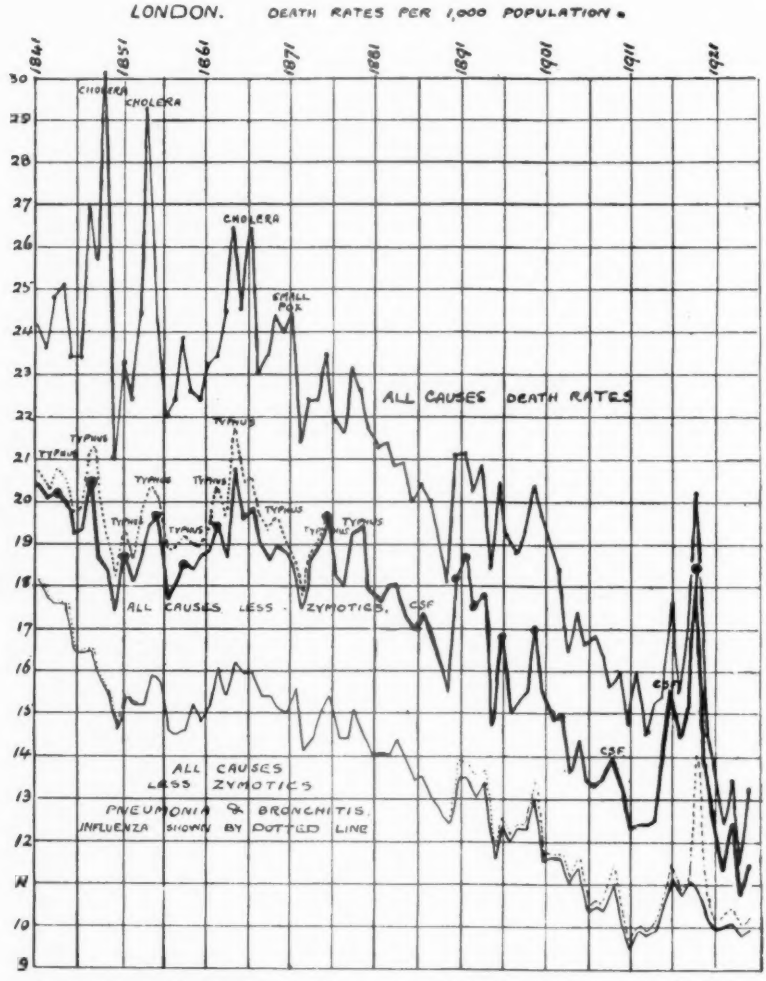


FIG. 3.—London death-rates in registration times illustrating the appearances of influenza on the crests of waves of fever.

that in this statement " he is quite correct, as the eruption in typhoid may occasionally simulate that of typhus so completely as to be indistinguishable from it." He refers to a patient of Sir William Jenner's, who " developed *typhoid* fever in the

ward and presented a *typhus* rash." As Stokes says, "there is no absolutely pathognomonic sign of any disease whatsoever" (Lectures, p. 234). The difficulty in distinguishing between typhus and cerebro-spinal fever is discussed by writers from the time of Stokes and Graves to Murchison. The last-named (p. 203) writes, "the diagnosis is not always so easy as may be imagined."

The conception "influenzal constitution" does not seem ever to have found favour with Jenner and Murchison, whose experience was acquired between the late forties and the end of the eighties. Sydenham says the lifetime of a physician is not long enough to follow the complete cycle of changes in epidemics. Dr. Greenwood, in 1913, observed that a scientific description of epidemic diseases presupposes: "(1) A disciplined and creative imagination, the mysterious spirit which the Hebrew poet conceived as moving over the face of the waters, fashioning therefrom an ordered creation: (2) a wide clinical experience, and (3) a considerable knowledge of contemporary and antecedent medical history." In the case of influenza it seems essential too that the writer's epidemiological experience be *not* limited to the trough between pandemics.

All epidemiologists are agreed that war, famine, crowding, etc., exercise vast influence upon "fevers." Graves has an interesting observation. He points out the close resemblance of the symptoms of starvation to those "observed in the worst forms of typhus," and adds, "it is not, I say, unreasonable to infer that gastric, cerebral and even pulmonary symptoms may supervene, analogous to those which result from actual starvation." Sydenham, in discussing his "new fever" of 1685 (Sched. Mon., par. 41 and 45), is disposed to ascribe petechiæ, purple spots, &c., to "malignity" and to faulty treatment. Farr's view is not at variance with this (Suppt. to 35th Ann. Rep., pp. 25-27). In a remarkable paragraph Jenner himself fairly sums up the situation. He says "the circumstances which favour the origin and spread of these diseases (his three continued fevers) are just the circumstances which favour the development of the most grave varieties of each; and, therefore, the development of those symptoms which render their general physiognomy the most nearly alike, low delirium, a black tongue, abundant sordes and extreme prostration, symptoms which at once arrested the attention of those who described epidemics in general terms. Again, petechiæ, as is well known, may occur in any disease in which the vital depression is extreme, e.g., malignant measles, small-pox, scarlatina; and the circumstances which promoted the origin and spread of epidemics in years gone by were just those which cause the disease to assume a low type, and, therefore, to be attended with petechiæ; and still further the circumstances in question are just those which favour the occurrence of dysentery as a complication of typhus; and as a consequence we find descriptions of a fever in which abundant eruption, frequent and bloody stools and great prostration were the most marked symptoms" (*loc. cit.*, p. 450). Have we not in these sentences the explanation of decline of typhus and relapsing fever, with apparent increase in mortality from influenza and pneumonia? There has been of late years actual decrease in bronchitis; and, despite the transfer of the fatal fevers with cerebro-spinal complication to the category of diseases of the nervous system, great reduction in mortality from diseases of the nervous system as a whole (see Ann. Rep. Med. Off., London, 1925).

The table facing p. 6 of the Ministry of Health Report gives the impression of ascending death-rates from influenza. But Farr's nomenclature obtained in the early period of registration, then Ogle's and then Tatham's. (Part III, Supplement, 75th Rep., pp. 48 and 231, shows the effect of these changes.)

The Registrar-General (10th Report, p. 42) estimated the excess deaths during nine influenza weeks, in five epidemics, as ranging from 8,071 (in 1733, the worst

epidemic) to 1,462 in 1837. In 1847 the corresponding figure was 6,066. He has since given later estimates, and has fully discussed methods of estimating, in his report on the epidemic of 1918-19. If, instead of merely taking deaths relating to the worst nine weeks, the mortality be computed over the five or six years covered by an "influenzal constitution," the excess death-rates in the prevalences of the nineties and those of recent years are distinctly less, in London, than in earlier experiences, despite the much greater concentration of population and the huge growth of intercommunication and traffic. Of course, the bettered conditions as regards living, hospital isolation, asylum provision, nursing and attendance upon the sick—and the important fact that resort is being made for medical aid, as time goes by, at less and less advanced stages of illness—are mainly responsible for this improvement.

Stress has been laid upon "fever" prevalence as the mark of "influenza in mufti," for the reason that it is the cases with marked fever that are apt to appear in death statistics; there is the further consideration that influenza with fever is perhaps especially infectious. It must not be inferred that in "endemic influenza," as a rule, fever is a predominant symptom. Peyton Blakiston, Bezly Thorne and Parsons all maintained that the primary attack in influenza was upon the central nervous system; Graves thought the vagus was particularly implicated, and Hecker held the same view as regards English sweat; while Smith Ely Jelliffe says "disturbance of the vagus is one of the most fundamental underlying conditions in influenza poisoning." In "endemic influenza" fever is often quite subordinated; a state of depression (with irritability and want of mental balance) might almost be regarded as the "pathognomonic symptom."

A practical lesson may be drawn from all this. While the graver complications associated with influenza now receive careful attention, the vastly more numerous mild attacks are still treated almost with derision, with the result that it is the usual practice for patients to move about freely among their fellows in the early infectious stages of illness. Moreover, many doctors are still "dissatisfied with the diagnosis, 'abdominal influenza,'" and there is crying need for education of public opinion regarding the infectious nature of the slighter (as well as of the more serious) respiratory and nervous manifestations of the disease. It should be plainly understood that there is a moral obligation upon all of us "to take our influenza lying down," and scrupulously to refrain, during the early infectious stages, from associating with others, using public conveyances, and going to places of public assembly. If this truth were fully realized, far more injury to the public health would be obviated than results at the present time from neglect of necessary precautions in *all* the "dangerous infectious diseases," among which, technically speaking, influenza is, as yet, *not* included.

[After the paper had been read Mr. LESLIE P. PUGH exhibited a number of slides illustrating the microscopical appearances in encephalitis lethargica in dogs (see *Lancet*, 1926 (ii), 950).]

Discussion.—Professor GREENWOOD remarked that he was becoming rather sceptical as to the importance of Sydenham's contribution to epidemiology; there seemed to him (Professor Greenwood) a good deal of point in Freind's almost contemporary criticism—"if we consider the method which this writer has adopted in curing these fevers, of, as he says, utterly different type, a method in which he was eminently successful, we shall find no trace of this distinction." (Freind's *Opera Omnia*, 1733, p. 238.) In fact, it did not appear that Sydenham's epidemiological *dicta* had any influence upon his actual practice.

He (Professor Greenwood) thought that Sir William Hamer had made two important contributions to epidemiological knowledge. The first, his characterization of the "setting" of an outburst of frank influenza, his cogent evidence of a relation in time between such an

outburst and, numerically, less important outbreaks of different illnesses especially those involving the nervous system. The second, his insistence upon taking a broad view and judging an epidemiological epoch as a whole. He thought, indeed, that Sir William pushed the method of historical comparison rather far. It was tempting, when one considered the vagaries of language—the same term used in several different senses and different words used to describe the same events—to generalize, even perhaps to wonder whether epidemiology as a whole were not a series of variations upon the theme of influenza. But it was possible to generalize so much that ultimately one reached the kind of oracular wisdom which, however true, was not helpful. It might be very unphilosophical to ask whether it were possible to determine a little in advance of the event when we were to have a frank outburst of what the man in the street called influenza, but one did ask it. The arithmeticians had certainly not provided any helpful answer and he did not really expect that any algebra, however ingenious, was going to produce a helpful answer from existing data. The philosophical epidemiologist might retort that, in a sense, we were *always* having epidemics of influenza; no doubt we were, but when were these epidemics going to be of such a type and magnitude that all of us, philosophers and others, would talk about "the epidemic of influenza"? In fact, as a prophet, the philosopher was no more helpful than the arithmetician, although he was more interesting. He (the speaker) believed that the most helpful attempt to answer the short-range question was likely to be that of the statistically-minded bacteriologist who systematically sampled the flora of the noses and throats of an adequate sample of apparently normal persons at regular intervals. Such work carried out in Manchester, by Professor Topley, at the instance of the Ministry of Health, gave distinctly encouraging results. There was a suggestion that before the actual clinical outburst of influenza there was a great increase in the rate of healthy pneumococcus-carriers. Should this be confirmed, an important step forward would have been taken.

Surgeon Commander S. F. DUDLEY said his experience of influenza in the Royal Navy confirmed Sir William's view that influenza was really always present in inter-epidemic periods. A ship might suffer from an outbreak involving a large percentage of the crew, months might pass without a case suggesting clinical influenza; then one or two cases of fever presenting the clinical characters seen in the original epidemic would suddenly appear, followed by no others. The influenza virus having fully exposed itself as an epidemic became submerged (in carriers) until some unknown stimulus caused it to just break surface again as an odd sporadic case or two. He did not go so far as Sir William in suggesting that definite bacteriological or clinical entities, such as cerebro-spinal fever or poliomyelitis, were due to the same biological agents, but he was convinced that the immutability of bacterial species (or preferably varieties) was less than orthodox bacteriological opinion allowed. Some viruses such as that of measles were very stable, and the epidemiological problems became simplified since the parasitic might be taken as a constant leaving the host and environment as two variables. In other conditions, most of all, he (the speaker) suspected, in influenza, the virus was very plastic, responding quickly to any change in the host and the environment (which included, of course, other bacteria). Variations in host resistance (herd-immunity), in the amount and nature of host-movement, and host-nutrition accounted probably for most of the periodic phenomena of epidemics, and all these factors might affect the distribution and characters of many distinct parasites at the same time.

Fleet Surgeon W. E. HOME, R.N., asked what was the significance of the terms "constitution," "epidemic constitution;" he said it was dangerous to use terms incompletely understood. The old eighteenth century physicians, whose works he had been lately reading, had no criteria for differentiating diseases, and their decisions as to the names of the diseases with which they were dealing were mere guesses. Thus Blane, even, had described the typhus cases he had seen on board ship about 1780 (in Rodney's time), noting that in some there were holes in the intestines, but surely these were cases of typhoid, not of typhus, and therefore his arguments about typhus were thus invalidated. It was along the lines of the essential differences of diseases, and the association of each with definite bacteriological causes, that we had been able to make such notable advances in the prevention of disease, the special aim of this Section, advances which would not otherwise have been secured.

Dr. E. W. GOODALL (in a communication by letter) stated that Sir William Hamer had apparently receded somewhat from the conception of the "constitution" which he held not

76 Hamer: *The Influenzal Constitution*

so very long ago. In the book entitled "Influenza," edited by Dr. Crookshank, and published in 1922, they were told that "by the term (epidemic constitution) we really mean to imply all the correlated symptomatic disorders and their consequences manifested in a community or population during a period of time that was not arbitrarily selected, but has natural duration and limitation, and during which prevalences and occurrences of distinctive nature arise." This did not appear to signify what Sydenham meant by the "epidemic constitution;" it embraced the idea of his "stationary fever" and the "inter-currents" which were subordinate to it, rather than the idea of the constitution itself, which was responsible for the stationary fever and set its stamp on the intercurrents. In respect of this definition Sir William, in the same book, allied himself with Dr. Crookshank; but both these authors "out-Sydenhamed" Sydenham when they actually wrote of, and endeavoured to explain, the *cause* of the constitution. However, they no longer, apparently, looked to extra-mundane agents or even to ultra-visible but terrestrial filter-passers for an explanation of the constitution and, therefore, of epidemics, but were content to remain in this world, where an "interplay of conflicting forces," such as "germs," "soils," "school influences," "mass immunity," and the like, were perhaps sufficient. As regards the phrase just quoted, "interplay of conflicting forces," he (Dr. Goodall) asked: Why conflicting? The various factors (he preferred this word to forces) might be by no means conflicting. They were much more likely to be aiding and abetting one another and the germs in the strife of the latter against unfortunate mortals.

He absolutely rejected the doctrine of the "epidemic constitution" as put forward by Sydenham, and so, apparently, did Sir William Hamer. The germ of the idea of Sydenham's "epidemic constitution," of the part it played in the establishment of the "stationary fever," and of the effects it had on the "intercurrents," was to be found in the writings of Galeazzo di Santa Sofia, published a century and a half before Sydenham wrote. It was curious that since Hecker gave us his "Epidemics of the Middle Ages" nearly a century ago, Galeazzo seemed to have been forgotten.

Was it not time to abandon the phrase "epidemic constitution"? Would it not be better to use instead the phrase coined by Peters in 1911, but with the extended meaning employed recently by Professor Topley, namely, "epidemic potential," "the balance of interacting forces which tends towards the occurrence of an outbreak of disease"?

Sir William Hamer, in accomplishing his laborious and difficult task, had taken Dr. Greenwood's advice and rejected that of Sir William Jenner. Why it should be absurd to try to settle the question of the identity of diseases by reference to old writers he failed to see. In respect of some diseases it might be very difficult, indeed even impossible, so to decide the question. One could rarely rely on the mere names of diseases as evidence of their presence in times long past; clear clinical descriptions were the only sure guide. Influenza was a particularly difficult disease to deal with. He was prepared to agree that the English sweating sickness was influenza, but not all the "agues" referred to by Creighton were. Sir William Hamer apparently suggested that much of what was called typhus was "influenza in mufti."

Sir William Hamer referred more than once to what he called the "setting" of influenza. Apparently the "setting" was not the same as the "constitution." It seemed to be the framework in which the epidemic, worked up by the potential, now appeared in respect of its relation to other epidemic diseases which might have appeared just before, or just after, or at the same time as itself. Otherwise he could not understand the frequent references to other diseases. It was useful, and indeed desirable, to know that when, for instance, there was a prevalence of encephalitis or cerebro-spinal fever, a severe epidemic of influenza was to be feared, or to be able to say that a severe epidemic of influenza might be followed by "trailers" of this, that, and the other character. That, however, did not much advance us in respect of what he took to be the most important business of the epidemiologist, namely to find out the particular factors, or forces, the interplay of which would bring about an epidemic of any particular disease. He looked for the solution of the problems of the "epidemic constitution" (again, with its new definition) more to the work carried on by the modern biometric school, as exemplified by the researches of Brownlee, Ross, Greenwood, Topley, Dudley and others, than to any searching amongst ancient medical writers.

Sir William Hamer asked: "Even if we had the minds of Sydenham and Willis, should we not still be considerably handicapped by the increased present-day complexity of interaction between germ and host?" He would answer with Sir William—"Yes, we should

be and we are." Epidemiological problems appeared to the great physicians of the past to be simpler of solution than they do to us. Sydenham thought he had to deal only with man and his environment, and when he found that his data were insufficient he invoked a ***deus ex machinâ***—the "epidemic constitution." He granted that Sydenham and Willis were ahead of their time, but did not admit that they were up to ours. He did not believe that Sydenham and Willis "intuitively discerned truths we find it hard to grasp." The truths they enunciated were established by sheer hard work, in the observation, recording and comparison of various facts, clinical and other, and their successors followed and were still following the same method.

Sir William Hamer devoted a considerable part of his paper to a consideration of certain questions connected with typhus. He stated that "'Typhus' cannot be regarded (speaking epidemiologically) as a clinical or pathological entity, distinguishable by a pathognomonic sign, mulberry rash, Weil-Felix reaction, or result of monkey inoculations." If Sir William Hamer would be willing to limit this statement to the period ending about the middle of last century he (Dr. Goodall) would agree with him to a very large extent. Since that time, however, typhus had been a perfectly distinct entity, epidemiologically as well as clinically. One could not deny epidemiological rank to a clinical entity which occurred so frequently and so widely throughout the world. Sir William referred to the so-called "tropical typhus" described by Fletcher and Lessler. He had not the faintest doubt that "tropical typhus" was the European typhus described by Jenner and Murchison. It was a very great pity that the disease had been already labelled "tropical typhus"—"typhus in the tropics," if preferred—but not "tropical typhus." The story was, in one way, the same as that of the so-called "Brill's disease," which was nothing but sporadic typhus. Sir William Hamer referred several times to the writings of Sir William Jenner, but he had read that great clinical physician to little purpose if he still had doubts of the entity of typhus fever.

Another statement about typhus was apparently intended to show that the "setting" of the influenza of 1799-1803 was a typhus setting. Now at that time typhus had not been separated from certain other fevers, and the word "typhus" was used with some vagueness. By one writer it would be employed to denote a definite fever, by another to signify a certain morbid state, in much the same way as we, at the present day, use the term "typhoid state." He had read again the chapter in Symes Thompson's book from which Sir William had made several extracts, and he thought that Sir William was misleading us, though doubtless, quite unintentionally, when he quoted Symes Thompson's statement that "it seemed to participate of typhus." As a matter of fact this statement was an extract made by his father, Theophilus Thompson, from an account given by a writer he did not name living at a place he did not mention, and that was very different from being Symes Thompson's summary. There was a very interesting section in the chapter from which these quotations were made which was headed "Relation of the Epidemic to other Diseases." There were fourteen of these diseases and the number of times each of them was associated with the influenza was as follows:—Scarlet fever, eight; measles, seven; typhus, three; tonsillitis and sore-throat, three; mumps, three; diarrhœa and cholera, two; and one each of some other diseases such as small-pox, whooping cough, and so on. This did not look to him much like a typhus "setting." Why should it not be called a scarlatinal or a morbillous setting?

He dissented from the statement that in measles the infecting power of the germ varied but little, and still more from the declaration that in that disease an incubation period of fourteen days might be regarded as constant.

He left Sir William Hamer to reconcile these inconsistencies. He submitted that the material of which the "setting" or framework of the influenzal constitution was composed was not altogether of the soundest.

He concluded by stating that Sir William Hamer's most interesting paper would set them all thinking, though they might not arrive at his conclusions. He (Sir William) appeared to have set out to make this particular ascent of the epidemiological Everest without the guide of clinical experience, so that he had, in consequence, long before he had reached a chimney or a slope—let alone the col—fallen into one of those crevasses which beset his way on the comparatively level, though dangerous, field of the glacier.

78 Hamer: *The Influenzal Constitution*

Sir WILLIAM HAMER (in reply) expressed his indebtedness to Dr. Chalmers for the teaching given in the lectures of two years ago; also to Dr. Greenwood for the paper on Sydenham of 1919, and for enlightenment on many points; and to Dr. Dudley for his recent studies on mass immunity, to which, as also to the closely related work of Greenwood and Topley on the same subject, he thought great importance must be attached. Fleet-Surgeon Home asked for a concise description of an "influenzal constitution" and he would refer him to that given in the essay on "The Theory of Influenza" in Crookshank's book published in 1922. Dr. Goodall's comments were most interesting. The endorsements coming from him, of some of the more important statements made in the paper, were especially valuable. He (Sir William) regretted he did not make it quite clear that Dr. Symes Thompson was giving the *ipsissima verba* of various writers. He remained, however, unrepentant as regards questioning the right to "epidemiological rank" of the "clinical entity, typhus."

Section of the History of Medicine.

President—Mr. WALTER G. SPENCER, O.B.E., M.S.

English Physicians—'Doctorati'—at the University of Padua in the 'Collegio Veneto Artista' (1617-1771).

By Professor EDGAR MORPURGO, Padua.

(*Communicated October*, 1926.)

I.—HISTORICAL NOTE.

THE 'COLLEGIO VENETO ARTISTA' OF THE UNIVERSITY OF PADUA (1617-1806).

FROM its beginning until the second half of the eighteenth century, the University of Padua (*Gymnasium Patavinum*) was always frequented by English students. Indeed, as early as the year 1331 we find in the 'Statuti Patavini' the English students distinguished from the Scottish, even though they formed one 'nation.'[1] In the year 1534 the *Natio Anglica* was made separate and distinguished from the *Natio Scotica*, though both belonged to the 'Corporation' named 'Ultramontana,' all the members of which were foreigners. In 1603 the two nations were once again united under the title of *Anglica*, and under this title were enrolled English, Scottish, and Irish. The Anglican nation had, like all other nations, its own counsellors (*Consiliarii*), its beadle (*Bidellus*), its secretary (*Notarius sive Cancellarius*); it possessed a library, and enjoyed special privileges. The counsellors had the right to place their ensigns on the walls of the court, of the balconies and of the 'Aula Magna' of the University building.

Of the two Universities dedicated to study in Padua, the University of Law (*Universitas Dominorum Juristarum*), and the University of Arts (*Universitas Dominorum Artistarum*), the former, the richer and more ancient, was much more frequented by English students than the latter. It was the University of Arts that included the students of medicine. It is worthy of note, however, and has formerly been mentioned by several writers,[2] that many British students attended both Universities simultaneously, as did many Germans. The Matriculation of the English Law students has already been published,[3] that of the English Art students is missing. Nevertheless, it is possible to obtain the names of many English doctors and students who were in Padua in the XVth, XVIth, and the beginning of the XVIIth centuries, from various documents existing in the Ancient Archives of the University, and in the Archives of the Bishopric, as for example, the following:—

XVTH CENTURY.

Magister GULIELMUS de Anglia quondam Petri, Doctor Artium, in Padua, 1405.[4]
D. GULIELMUS HATKLIFF, Doctor Artium (1446).

[1] Brown, H. F., Inglesi e Scozzesi all 'Universita' di Padova dall' anno 1618 sino al 1765. In *Monografie Storiche sullo Studio di Padova. Contributo del R. Instituto Veneto di Scienze lettere ed arti alla celebrazione del VII Centenario della Universita.* Venice, 1922; p. 137.

[2] As for example: Favaro, A., *Galileo Galilei e lo Studio di Padova*, Florence, 1883, and *Amici e corrispondenti di Galileo Galilei*; XXVI, Giovanni Wedderburn; XXVII, Riccardo White; XXVIII, Riccardo Willougby.

[3] J. A. Andrich, *De natione Anglica et Scota Juristarum Universitatis Patavinae, ab anno MCCXXII post Chr. n. usque ad a. MDCCXXXVII*, Padua, 1892.

[4] Gloria, A., *Monumenti della Universita' di Padova* (1318-1405), Tome 1, Padua, 1888.

64 Morpurgo: *English Physicians at the University of Padua*

D. ROBERTUS FLEMMING, Doctor Artium et Bacc. Theologiae (1442).[1]
D. GULIELMUS, De Anglia (Guglielmo d'Inghilterra), Doctor Artium (1447).[2]
D. THOMAS LINACRE (1492).

XVITH CENTURY.

D. EDUARDUS BROWN, Anglus, Doctor Artium (6 Aprilis, 1525).
D. THOMAS BILL, Anglus, D. Joannis filius (8 Julij, 1533).
D. JOANNES FRIER, Anglus (21 Januarij, 1536).
D. JOANNES CAJUS, D. Roberti filius, Britannus (3 Majj, 1541).
D. THOMAS TWINE, D. Joannis filius (29 Junij, 1570).[3]

XVIITH CENTURY.

D. WILLIAM HARVEY (1602).
D. JOSEPH NOBBE, Anglus, D. Roberti filius (23 Aprilis, 1603).
D. JOANNES MORE, Anglus, qm Odoardi filius (7 Septembris, 1605).
D. JOANNES FRIER, Anglus, D. Thomae filius (6 Aprilis, 1610).
D. JOANNES WEDDERBURN, Scotus (12 Augusti, 1612).
And many others.[4]

On the Roll of the Royal College of Physicians of London are also several names of Doctors in Medicine who took their degree at Padua, as D. JOH. CHAMBRE (1502), D. EDWARD WORTON (1522), D. CESAR A. DALMARIS (1545), D. GEORGE TURNER (1582), D. EDWARD JORDAN (1591), D. GEORGE ROGERS (1612), D. PAUL DE LAUNE (1614), D. ELEAZAR HODSON (1616), etc.[5] Other names are mentioned by G. H. Darwin.[6]

The conferring of the degree on the Arts students (Philosophers, Med. Doctors, and Theologians) took place in the same way as that of the Law students, with great solemnity in the presence of the Bishop, Grand Chancellor of the University and of the 'Sacred College of Philosophers and Doctors.'[7] During the time of the Reformation, Pope Pius IVth (De Medici) in a Bull dated 13th November, 1564 ('In sacrosancta'), enjoined upon those taking a degree an oath of profession of the Catholic Faith ('*Professio fidei*').[8] Those who did not belong to this religion received their degree from the Counts Palatine (Comites Palatini), but as this system gave rise to many difficulties, the Government of the Venetian Republic, following the advice of the celebrated Councillor Fra Paolo Sarpi, instituted for non-Catholics, by Decree dated 22nd April, 1616, the Venetian College of Arts (*Collegio Veneto Artista*). In this College those students who did not profess the Catholic faith received the '*laurea*' in philosophy and medicine, and in philosophy or in medicine only, or the '*licentia*' in surgery '*auctoritate veneta.*' In 1635, 'Franciscus Erizzo Dux Venetiarum' instituted the *Collegio Veneto Giurista* for the Law students who did not profess the Catholic faith as well.

The names of the '*Doctorati*' by the Counts Palatine are almost entirely missing, whereas those '*Doctorati*' in the Venetian College of Arts, from 1617 to 1806, are still almost all ascertainable. There is a brief period from 1648 to 1652 in which the names of students taking degrees in philosophy and medicine are missing; the names of those qualified in surgery during this period are, however, available.

1 G. Zonta and G. Brotto, *Acta Graduum Academicorum Gymnasii Patavini ab Anno MCCCCVI ad Annum MCCCCL cum aliis antiquaribus in Appendice additus iudicio historico collecta ac digesta curantibus*, Padua, 1922.

2 Royal Library of the University of Padua: Cod. Man. 43; A. F. Dorighello, *Elenco di Laureati in Padova sec. XIV-XVIII.*

3 Dorighello, II, 292.

4 Brown, H. F., Inglesi, Scozzesi all 'Universita' di Padova, etc.

5 W. Munk, *The Roll of the Royal College of Physicians of London.* London, 1878.

6 G. H. Darwin, *On monuments to Cambridge men at the University of Padua.*

7 G. Giomo, *L'Archivio antico della Universita di Padova*, Venice, 1893.

8 B. Brugi and I. A. Andrich, *loc. cit.*

The 'Collegio Veneto Artista' was actually in existence from 1617 to 1797; in the latter year the name was altered to 'Collegio Nazionale.'[1] In 1798 the Collegio again assumed its former title of Collegio Veneto, until the year 1806, that is, it was retained until the abolition of the College by the Napoleonic reforms. Originally the Collegio Veneto Artista was composed of the professors holding the chairs of philosophy, theoretical and practical medicine, and other doctors. Later, the number of professors was increased to eight, one of the oldest or most learned was elected President of the College, remained in office for three years, and could be re-elected. In his absence his place was taken by one of the principal professors of theoretical and practical medicine. At the ceremony of the conferring of degrees, which was held in public, there were present the Rector or the Pro-rector, or the Counsellors of the University of Arts, teachers, doctors from the city, scholars, etc. The Nobles, Clergy, and those who had taken other degrees, were qualified *more nobilium*, that is to say, they were exempt from an oral examination.

In order to obtain a degree in the Collegio Veneto Artista the candidates were obliged to pay:—

For the ordinary degree in philosophy and medicine	Venetian lire :	886 : 01
For the degree *more nobilium* in philosophy and medicine, including sweets and gloves	Venetian lire :	1395 : 09
For the ordinary degree in surgery	*latine sermone* :	500 : 15
For the ordinary degree in surgery	*vulgari sermone* :	232 : 02
For the degree in philosophy and medicine for Jews, including sugar, and sweets	Venetian lire :	1640 : 01
For the degree in surgery	*latine sermone* for Jews, lire :	836 : 15
For the degree in surgery for Jews, lire, including sugar	*vulgari sermone* :	298 : 02

After the foundation of the Collegio Veneto Artista, the English Catholics continued to take their degree before the Bishops and the '*Sacred College of Philosophers and Doctors* as in the past, at the same time making a profession of Faith.'

I have collected the names of the English students 'Doctorati in almo Collegio Veneto DD. Artistarum' from the foundation in 1617 to 1771, after which year I find no more British names. This task was very often difficult on account of the changes effected in the English names in the Latin documents drawn up by Chancellors and Beadles, who were completely ignorant of the English language. In some cases the names were written as dictated by the students themselves, and so were spelled as pronounced, in other cases the names were crudely transformed. It will be interesting to cite several examples:—

ENGLISH.	LATIN.
THOMAS CADEMAN	THOMAS GADMAN
WILLIAM WISEMAN	GULIELMUS ZBISSIMAN
JOHN MICKLETHWAYT	JOANNES MICHELSUGIONIT
JOSEPH COLSTON	JOSEPHUS COLTON
JAMES WILLIMOT	JACOBUS ZWILLIMOTTUS
THEODORE DE VAUX	THEODORUS DE VALLIBUS
THOMAS LAWRENCE	THOMAS LAURENTIUS
WILLIAM MORETON GILKES	GULIELMUS MORTON GLIKES

In the reconstruction of the various names, a task which has required patience, I have in many cases made use of the works of various English authors: W. Munk, G. H. Darwin, H. F. Brown. The several variations of the same surname found in the different books and registers are written in parenthesis. The Roll is compiled from the Ancient Archives of the Royal University of Padua (Archivio Antico della R. Universita' di Padova).

1 '*Archivio Antico della R. Universita*,' Padua, No. 294.

66 Morpurgo: *English Physicians at the University of Padua*

II.—ANGLI 'LAUREATI' IN PHILOSOPHIA ET MEDICINA IN COLLEGIO VENETO, ALMAE UNIVERSITATIS DD. ARTISTARUM, PADUAE. (MDCXVII—MDCCLXXI POST CHR. NATUM.)

EXCERPTA EX ANTIQUO TABULARIO.

ALMI GYMNASII PATAVINI.

Volumina 274-295: *Doctores Philosophiae et Medicinae itemque Licentiati in Chirurgia in Collegio Veneto Almae Universitatis DD. Artistarum ab Anno MDCXVII usque ad Annum MDCCCVI, Post Chr. Natum.*

Anno

1. 1618, Indictione I, die Sabbati 27 mensis Januarij, hora 21 in circa, in Schola Magna celeberrimi Patavini Gymnasii:
   D. LAURENTIUS WRIGHT, Romfordiensis, Anglus, lauream in Medicina consecutus est.
2. 1619, Indictione II, die Lunae 16 mensis Septembris, hora 21 in c.:
   D. PETRUS CHAMBERLANUS (alias CHAMBERLAN), Anglus, Eximi D. Petrifilius, lauream in Philosophia et Medicina consecutus est.
3. 1620, Indictione III, die Jovis 19 mensis Martij, hora 14 inc.:
   D. THOMAS GADMAN (alias CADEMAN, CADYMAN, GADMEN), Anglus, Horfieldensis, D. Thomae filius, lauream in Philosophia et Medicina consecutus est.
4. 1620, Indictione III, die Veneris 24 mensis Aprilis, hora 21 in circa:
   D. RICHARDUS BERIE (alias BERRY), Anglus, filius quondam Thomae, lauream in Philosophia et Medicina consecutus est.
5. 1621, Indictione IV, die Mercurij 10 mensis Novembris, hora 22 inc.:
   D. GULIELMUS GORDONUS, Aberdonensis, Scotus, D. Georgij filius, lauream in Philosophia et Medicina consecutus est.
6. 1622, Indictione V, diebus 13 et 15 Mensis Januarij:
   D. JOANNES BASTWICUS, Anglus, lauream in Phil. et Med. consecutus est.
7. 1623, Indictione VI, die Lunae 2 Octobris, hora 21 in circa:
   D. JOANNES BROUNS (alias BROWNE), Anglus, D. Joannis filius, lauream in Medicina tantum, consecutus est.
8. 1627, Indictione IX, die Lunae 15 mensis Februarij, hora 17 in circa:
   D. COSTANTINUS ROBERTSONUS, Scotus, lauream in Phil. et Med. consecutus est.
9. 1627, Indictione IX, die Veneris 19 mensis Martij, hora 21 in circa:
   D. THOMAS D'ALBRET, Scotus, Edimburgensis, D. Valentini filius, lauream in Phil. et Med. consecutus est.
10. 1627, Indictione I, die Lunae 5 mensis Julij, hora 12 inc.:
    D. HUGO LAUTENS, Scotus, Edimburgensis, D. Davidis filius, lauream in Phil. et Med. consecutus est.:
11. D. JACOBUS HAYMUS (HAYUS ?), Scotus, D. Gualterij filius, lauream in Phil. et Med. cons. est.
12. 1627, Indictione I, die Mercurij 14 mensis Julij, hora 12 inc.:
    D. GULIELMUS ZBISSIMAN [*sic*], (alias WISEMAN), Anglus, D. Thomae filius, et.:
13. D. GULIELMUS GODDARD, Anglus, D. Thomae fil. lauream in Philosophia et Medicina consecuti sunt.
14. 1627, Indictione VII, die Jovis 19 Augusti, hora 12 in circa:
    D. EDMUNDUS RANDULPHUS, Anglus, lauream in Phil. et Med. consecutus est, et:
15. D. PHILIPPUS BARSONIUS, Anglus, D. Richardi filius, lauream in Medicina tantum consecutus est.
16. 1628, Indictione XI, die Martis 2 mensis Majj, hora 13 in circa:
    D. MAURITIUS WILLIAMUS, (alias WILLIAMS), Anglus, Londinensis, D. Ludovici filius, lauream in Philosophia et Medicina consecutus est.
17. 1628, Indictione XI, die Lunae 4 mensis Decembris, hora 22 inc.:
    D. JOANNES LOUNDE, (alias LOUNDS), Anglus, D. Gulielmi filius, lauream in Phil. et Med. cons. est.

Anno

18. 1629, Indictione XII, die Lunae 25 mensis Junij, hora 22 inc.:
D. THOMAS HEIGHAM, (alias HIGHAM, HEINGHAM), Anglus, D. Joannis filius, lauream in Philosophia et Medicina consecutus est.

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19. 1630, Indictione XIII, die Lunae 4 Februarij, hora 21 in circa:
D. JOANNES KIRTRONUS (alias KIRTON, KIRTROMS), Anglus, lauream in Philosophia et Medicina consecutus est.

20. 1630, Indictione XIII, die Mercurij 5 mensis Junij, hora 22 inc.:
D. ROBERTUS BODIUS, Scotus, Aberdonensis, D. Archibaldi filius, lauream in Philosophia et Medicina consecutus est.

21. 1632, Indictione XV, die Martis 12 mensis Octobris, hora 16 inc.:
D. RICHARDUS HUNTON (HANTON ?), Lincolniensis, Anglus, q.m. D. Joannis filius, lauream in Phil. et Med. consecutus est.

22. 1632, Indictione XV, die Mercurij 27 mensis Octobris, hora 22 inc.:
D. THOMAS NOTT, Anglus, D. Rugerij filius, lauream in Phil. et Med. cons. est.

23. 1634, Indictione II, die Veneris 24 mensis Novembris, hora 16 inc.
D. PHILIPPUS VINCENTIUS FIRSBAEUS (alias FYRSBEUS), Eboracensis, Anglo-britannus, D. Gerardi (alias Ricardi) filius, lauream in Medicina tantum consecutus est.

24. 1635, Indictione III, die Veneris 27 mensis Julij, hora 20 inc.:
D. JACOBUS BETTON, D. Jacobi filius, Scotus, Angussonus, lauream in Phil. et Med. cons. est.

25. 1635, Indictione III, die Mercurij 12 mensis Octobris:
D. FRANCISCUS HEVSE (HOWSE ?), Anglus, D. Nicolai filius, lauream in Phil. et Med. cons. est.

26. 1635, Indictione III, die Veneris 21 Novembris, hora 21 inc.:
D. THOMAS WESTBY (alias WESBE), Anglicus [*sic*], lauream in Phil. et Med. cons. est.

27. 1635, Indictione III, die Sabbati 17 mensis Decembris, hora 22 inc.:
D. GULIELMUS GRENS, Anglus, lauream in Philosophia et Medicina consecutus est.

28. 1636, Indictione IV, die Martis 21 mensis Februarij:
D. THADEUS CARPENIS [*sic*], Lugdunus, D. Vibaldi filius, lauream in Phil. et Med. consecutus est.

29. 1636, Indictione IV, die Lunae 13 mensis Martij, hora 18 inc.:
D. ENDRICUS VALDANEA [*sic*], Anglus, '*Pro maiori parte*,' lauream in Philosophia et Medicina consecutus est.

30. 1636, Indictione IV, die Jovis 10 mensis Aprilis, hora 20 inc.:
D. SAMUEL REMINTON (alias REMINGTON), Anglus, q.m. D. Danielis filius, lauream in Medicina tantum consecutus est, et postea:

31. D. ANTONIUS HAVIT (alias HEWETT), Anglus, D. Joannis filius, lauream in Philosophia et Medicina consecutus est.

32. 1636, Indictione IV, die Lunae 28 mensis Aprilis, hora 13 inc.:
D. GEORGIUS HENT, Anglus, D. Thobiae filius, lauream in Phil. et Med. consecutus est.

33. 1637, Indictione I, die Sabbati 11 mensis Julij, hora 21 inc.:
D. ENDRICIUS STAMLHEI (alias STANLEY), Anglus, D. Endrici filius, lauream in Medicina tantum consecutus est, et postea:

34. D. JOANNES HOUGHAM (HOUGHTON ?), Anglus, D. Rugerij filius, lauream in Medicina tantum consecutus est.

35. 1638, Indictione VI, die Sabbati 17 mensis Julij, hora 12 inc.:
D. JOANNES WARDER (alias VARDE'), Anglus, D. Danielis filius, et:

36. D. THOMAS CHARLET (alias CHARLAST), Anglus, D. Francisci filius, lauream in Medicina tantum consecuti sunt.

68 Morpurgo: *English Physicians at the University of Padua*

Anno

37. 1638, Indictione VI, die Sabbati 14 mensis Augusti, hora 12 inc.:
D. ROBERTUS CHILD, Anglus, D. Joannis filius, lauream in Medicina tantum consecutus est, et postea :

38. D. THOMAS SOMERVELLIUS, Scotus, D. Joannis filius, lauream in Phil. et Med. consecutus est.

39. 1638, Indictione VI, die Martis 30 mensis Decembris, hora 17 inc.:
D. JOANNES MICHELSUGIONIT [*sic*] (alias MICKLETHWAYT), Anglus, D. Thomae filius, lauream in Medicina tantum consecutus est.

40. 1639, Indictione VII, die Veneris 26 mensis Augusti, hora 12:
D. LEVINUS FLUD (alias FLUDD), Gensiensis [*sic*], Anglus, D. Thomasij filius, lauream in Medicina tantum consecutus est.

41. 1640, Indictione VIII, die 2 mensis Octobris, hora 14 inc.:
D. RICHARDUS LONDON, Anglus Norfolcensis, lauream in Medicina tantum consecutus est.

42. 1640, Indictione VIII, die 6 mensis Decembris, hora 18 inc.:
D. JOANNES SAVIT, Bedfordiensis, Anglus, D. Oliverii filius, lauream in Phil. et Med. cons. est.

43. 1640, Indictione VIII, die Mercurii 12 mensis Decembris, hora 18 inc.:
D. JOANNES DE AULIS, Blandfordensis, Anglus, D. Gulielmi filius, lauream in Phil. et Med. consecutus est.

44. 1641, Indictione IX, die 1 mensis Martij, hora 16 in circa:
D. EDUARDUS VALDRON, Londinensis, Anglus, lauream in Phil. et Med. cons. est.

45. 1641, Indictione IX, die Martis 10 mensis Septembris, hora 13 inc.:
D. JOANNES HALEUS (alias HALES), Anglus, D. Richardi filius, lauream in Phil. et Med. cons. est.

46. 1641, Indictione IX, die Veneris, 13 mensis Decembris, hora 17 inc.:
D. THOMAS COXE', Anglus, ex agro Somersha [*sic*] (alias Somersettensis), D. Thomae filius, lauream in Philosophia et Medicina consecutus est.

47. 1642, Indictione X, die Lunae 17 mensis Februarij, hora 16 inc. :
D. ALLANUS FOSTERUS, Anglolondinus, D. Gulielmi Equitis Aureati filius, lauream in Philosophia et Medicina consecutus est.

48. 1642, Indictione X, die Mercurij 26 mensis Februarij, hora 15 inc.:
D. JOSEPHUS DEY, Anglonorricensis, D. Roberti filius, lauream in Medicina tantum consecutus est.

49. 1642, Indictione X, die Martis 13 mensis Majj, hora 14 inc.:
D. NATHANAEL ANDREWES (alias ANDREWS), Londinensis, Anglus, D. Thomae filius, lauream in Phil. et Med. consecutus est.

50. 1642, Indictione X, die Jovis 28 mensis Augusti, hora 13 inc.:
D. JACOBUS GIBBESIUS (GIBBES), Londinensis, Anglus, D. Gulielmi filius, lauream in Phil. et Med. consecutus est.

51. 1642, Indictione X, die Sabbati 30 Augusti, hora 13 in circa:
D. STEPHANUS TAYLERUS, Anglus, D. Stephani filius, lauream in Philosophia et Medicina consecutus est.

52. 1642, Indictione X, die Mercurij 15 mensis Octobris, hora 13 inc.:
D. MATHEUS BACONUS, Anglus Norfolcensis, Nob. D. Richardi filius, lauream in Philosophia et Medicina consecutus est.

53. 1642, Indictione X, die Mercurij 31 mensis Decembris, hora 17 in circa:
D. JOSEPHUS COLTON (alias COLSTON, COLESTON), Anglus, D. Joannis filius, lauream in Medicina tantum consecutus est.

54. 1643, Indictione XI, die Jovis 31 mensis Decembris, hora 17 inc.:
D. JOANNES POERUS, Clonmelliensis, Ibernus, D. Georgij filius, lauream in Philosophia et Medicina consecutus est.

55. 1644, Indictione XII, die Martis 12 mensis Januarij, hora 21 inc.:
D. GULIELMUS HAWESIUS, Bedfordiensis, Anglus, D. Thomae filius, lauream in Medicina tantum consecutus est.

56. 1644, Indictione XII, die Sabbati 21 mensis Majj, hora 12 inc.:
D. THOMAS BRANDONIUS, Londinensis, Anglus, D. Thomae filius, lauream in Medicina tantum consecutus est.

Anno

57. 1644, Indictione XII, die Sabbati 21 mensis Majj:
D. DEMETRIUS HARNIUS, Hibernus, D. Demetrii filius, lauream in Phil. et Med. cons. est.

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58. 1645, Indictione XIII, die Sabbati 15 mensis Aprilis, hora 21 inc.:
D. ROBERTUS DAULINUS, Scotus, lauream in Medicina tantum consecutus est.

59. 1645, Indictione XIII, die Mercurij 31 mensis Majj, hora 11 inc.:
D. RICHARDUS GIBBON, Anglus, D. Thomae filius, lauream in Phil. et Med. cons. est.

60. 1645, Indictione XIII, die Jovis 17 Augusti, hora 12 inc.:
D. JACOBUS ZWILLIMOTTUS (alias WILLIMOT), Anglus, D. Jacobi filius, lauream in Medicina tantum cons. est.

61. 1645, Indictione XIII, die Veneris 27 mensis Octobris, hora 15 inc.:
D. GEORGIUS NISBET, Scotus, D. Georgij filius, lauream in Philosophia et Medicina consecutus est.

62. 1646, Indictione XIV, die Lunae 30 mensis Aprilis, hora 12 in circa:
D. GEORGIUS ROGERS, Anglus, D. Georgij filius, lauream in Medicina consecutus est.

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63. 1652, Indictione V, die Jovis 31 mensis Octobris, hora 16 in circa:
D. ALEXIUS VODEKA' (alias VODKA), Anglus, Eboracensis, D. Comitis Alexij filius, et:

64. D. GEORGIUS BEARE, Devoniensis, Anglus, D. Georgij filius, lauream in Philosophia et Medicina consecuti sunt.

65. 1652, Indictione V, die Lunae 23 mensis Decembris, hora 17 inc:
D. JOANNES PIERS, Hibernus, Clanen sis, D. Nicolai filius, lauream in Phil. et Med. cons. est.

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66. 1655, Indictione VIII, die Veneris 8 Octobris:
D. GUALTERUS JAMESIUS, Anglus, D. Gualteri filius, lauream in Phil. et Med. consecutus est.

67. 1655, Indictione VIII, die Jovis 21 Octobris:
D. PETRUS VAVASOUR, Anglus, Eboracensis, D. Equitis Thomae filius, lauream in Medicina tantum, consecutus est, et:

68. D. JOANNES MORTON, Cambrio-britanus, Flintsariensis, D. Joannis filius, lauream in Phil. et Med. consecutus est.

69. 1655, Indictione VIII, die 30 Octobris:
D. TEODORUS DE VALLIBUS (alias DE VAUX), Anglus, D. Thomae filius, lauream '*Pro maiori parte*,' in Medicina tantum, consecutus est.

70. 1656, Indictione IX, die Veneris 27 Julij, hora 12 inc.:
D. HENRICUS TICHBURNE (alias TICHBORNE), Anglus, D. Michaelis filius, lauream in Philosophia et Medicina consecutus est.

71. 1656, Indictione IX, die Jovis 3 Augusti, hora 12 inc.:
D. RICHARDUS HERINGTONUS (alias ERINGTONUS), Anglus, D. Richardi filius, lauream in Phil. et Med. cum gratia consecutus est.

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72. 1657, Indictione X, die Lunae 31 mensis Decembris, hora 16 inc:
D. ROBERTUS LION, Anglus, D. Petri filius et:

73. D. ANDREAS BEECH, Anglus, D. Andreae filius, lauream in Medicina tantum consecuti sunt.

74. 1658, Indictione X, die mensis Februarij, mane:
D. THOMAS LAURENTIUS (alias LAWRENCE), Anglus, D. Joannis filius, lauream in Medicina tantum consecutus est.

Anno

75. 1658, Indictione X, die 4 Februarij, mane:
D. JACOBUS PARAVICINUS, Anglus, D. Petri filius, lauream in Philosophia et Medicina consecutus est, et:

76. D. GULIELMUS PARKER, Anglus, D. Petri filius, lauream in Medicina tantum consecutus est.

77. 1658, Indictione XI, die Veneris 29 Martij, mane:
D. GULIELMUS BRIGHT (alias WRIGHT), Anglus, D. Enrici filius, lauream in Phil. et Med. consecutus est.

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78. 1658, Indictione XI, die 6 mensis Decembris, hora 16 inc
D. PHILIPPUS BRATHWAIT, Anglus, D. Richardi filius, lauream in Phil. et Med. cons. est.

79. 1658, Indictione XI, die Lunae 23 mensis Decembris, hora 17 inc:
D. GEORGIUS SMITH, Anglus, q.m. D. Joannis filius, lauream in Medicina tantum consecutus est, et postea:

80. D. RICHARDUS TREVOR, Anglus, q.m D. Joannis filius, lauream in Medicina tantum consecutus est, habita fide quod praedicti doctorati fuerunt solum modo in Philosophia in Alma Academia Oxoniensi a Ex. mo D. Doctore Thomasio Laurentio Anglo et a Domine Thomasio More Anglo.

81. 1659, Indictione XII, die 1 mensis Februarij, hora 16 inc.:
D. JOANNES LAWSON, Anglus, q.m. D. Rhoberti filius, lauream in Medicina tantum consecutus est, quia privilegium suum Philosophiae praesentaverat ex Alma Academia Cantambrigiensi datum die 3 Januarij, 1657.

82. 1659, Indictione XII, die Veneris 14 mensis Martij, hora 16 inc.:
D. GULIELMUS VALGRAVICUS (WALDEGRAVICUS), Anglus, D. Enrici filius, lauream in Philosophia et Medicina consecutus est.

83. 1659, Indictione XII, die Lunae 16 mensis Augusti, hora 14 inc.:
D. THOMAS MORUS (alias MORE), Anglus, D. Samuelis filius, lauream in Medicina tantum consecutus est.

84. 1660, Indictione XIII, die Jovis 30 mensis Decembris, hora 18 inc.:
D. PETRUS BALLE' (alias BALLE), Anglus, D. Petri filius, lauream in Philosophia et Medicina consecutus est.

85. 1661, Indictione XIV, die Jovis 12 mensis Majj, hora 14 in circa:
D. THOMAS HARPOM (alias HARPAUN), D. Odoardi filius, Anglus, Londinensis, lauream in Phil. et Med. cons. est.

86. 1661, Indictione XIV, die Veneris 29 mensis Julij, hora 13 in circa:
D. CAROLUS CONQUISTUS (alias CONGUESTUS), Anglus, Londinensis, D. Richardi filius, lauream in Phil. et Med. consecutus est.

87. 1661, Indictione XIV, die Martis 30 mensis Augusti, hora 14 in c.:
D. WILLIAM STOKHAM (alias STOKEHAM), Anglus, D. Gulielmi filius, lauream in Phil. et Med. consecutus est, ad praesentiam D. Roberti Naperij Nob. Angli, Almae Universitatis DD Artistarum Dig. mi Sindici et Pro-Rectoris.

88. 1662, Indictione XV, die Martis 29 Augusti, hora 13 in circa:
D. ROBERTUS NAPIERUS (NAPIER), Anglus, Lintonensis, Ex. D. Nob. Roberti filius, *more nobilium*, lauream in Philosophia et Medicina consecutus est.

89. 1662, Indictione XV, die Lunae 5 mensis Junij, hora 12 in circa:
D. FRANCISCUS HEMSTERHICIS (?), q.m. D. Tiberij, Scotiae, Frisus, lauream in Phil. et Med. cons. est.

90. 1664. Die Sabbati 27 mensis Septembris, hora 15 in circa:
D. RICHARDUS DRAPERUS (DRAPER), Anglus, D. Richardi filius, lauream in Medicina tantum consecutus est, et postea:

91. 1664, D. GABRIEL HOWIFIELD (alias HONYFIELD), Anglus, D. Richardi filius, lauream in Philosophia et Medicina consecutus est.

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92. 1665, Die Martis 15 mensis Septembris, hora 13 in circa:
D. THOMAS DOUGLASSIUS (DOUGLASS) D. Andreae filius, Scotus, lauream in Medicina tantum consecutus est, quia privilegium suum Philosophiae praesentavit ex Alma Academia Edinburgi, die 27 Juni, 1655, et postea:

Section of the History of Medicine 71

Anno

93. D. GULIELMUS CLERK q.m. D. Georgij, Scotus, lauream in Medicina tantum, consecutus est.

94. 1666, Die Mercurij 14 mensis Aprilis:
D. HUGONUS KENNEDUS, q.m. D. Alinerij, Scotus, lauream in Phil. et Med. consecutus est.

95. 1668, Die Mercurij 6 mensis Septembris, hora 13 in circa:
D. EZECHIEL TANNER, Anglus, q.m. D. Ezechielis filius, lauream in Phil. et Med. cons. est.

96. 1669, Die Veneris 29 mensis Martij, hora 18 in circa:
D. JACOBUS BORTHVEICHUS (alias BORTHVICUS), Scotus, Harlowiensis, D. Jacobi filius, lauream in Phil. et Med. cons. est.

97. 1670, Die Mercurij 2 mensis Aprilis, hora 13 in circa:
D. ROBERTUS WARDE' (WARD), Anglus, D. Odoardi filius, lauream in Phil. et Med. cons. est.

98. 1670, Die Lunae 7 mensis Julij, hora 11 in circa:
D. GULIELMUS STAHNLEY (STANLEY), Anglus, Londinensis, D. Joannis filius, lauream in Phil. et. Med. consecutus est.

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99. 1674, Die Veneris 5 mensis Octobris, hora 16 in circa:
D. RUBERTUS BANNERMANN, Scotus, Aberdonenensis, q.m. D. Ruberti filius, lauream in Phil. et Med. consecutus est.

100. 1675, Die Veneris 23 mensis Augusti, hora 13 in circa:
D. CHRISTOPHORUS JRVIN, Scotus, Edimburgensis, D. Christophori filius, lauream in Phil. et Med. consecutus est.

101. 1677, Die Sabbati 8 mensis Majj, hora 13 in circa:
D. ALEXANDER CRAVSTON, Scotus, D. Thomae fil., lauream in Medicina tantum, consecutus est, quia privilegium suum Philosophiae presentavit ex Academia Edimburgensi anno Domini 1674.

102. 1677, Die Mercurij 26 mensis Majj, hora 13 in circa:
D. JOANNES HUTTON, Scotus, D. Joannis filius, lauream in Philosophia et Medicina consecutus est, et post prandium, hora 20 in circa:

103. D. JOANNES MARGILL (MAKGILL), Scotus, D. Jacobi filius, lauream in Philosophia et Medicina consecutus est.

104. 1677, Die Martis 7 mensis Novembris, hora 17 in circa:
D. PATRITIUS CAMERARIUS, Scotus, D. Gulielmi filius, lauream in Medicina tantum consecutus est cum in Philosophia gradum accepisset in Universitate Regia Aberdenensi, habita fide EE.DD. Jacobi Cadenedi Scoti, Professoris et Alexandri Crawston Scoti, Doctoris.

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105. 1681, Indictione IV, die Jovis 12 mensis Junij, hora 12 in c.:
D. JOANNES WATSONUS, Scotus, D. Alexandri filius, lauream in Phil. et Med. consecutus est.

106. 1681, Indictione IV, die Mercurij 9 mensis Julij, hora 12 in circa:
D. EDMUNDUS DAVIUS (DAVIE), Anglus, D. Hunfrij filius, lauream in Medicina tantum consecutus est, quia privilegium suum Philosophiae presentavit ex Alma Academia Cantabrigensi (circiter Anno 1676) habita fide etiam cum juramento a Ex-D. Joanne Watson Scoto, filio D. Alexandri et a D. Gulielmo Neighbour, filio D. Gulielmi Scoto.

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107. 1682, Indictione V, die Sabbati 19 mensis Septembris, hora 15 in circa:
D. JOANNES ROTTWE' (alias ROWE), Anglus, D. Georgij filius, lauream in Philosophia et Medicina consecutus est, et postea:

108. D. DANIEL CALLAGHAM, Hibernus, q.m. D. Joannis, filius lauream in Medicina tantum, consecutus est.

Anno

109. 1682, Indictione V, die Sabbati 3 mensis Octobris, hora 14 in c.:
"Eruditus Vir" D. JOANNES HUNGERFORD, Oxoniensis, Anglus, D. Francisci filius, *Artium Magister* è Collegio Corp. Christi Oxoniae, lauream in Medicina tantum, consecutus est, quia lauream philosophicam acceperat in Universitate Oxoniensi, ut affirmarunt cum juramente Ex. D.D. Daniel Callagham, Hiberniensis, et Joannes Rowe', Anglus.

110. 1683, Indictione VI, die Jovis 4 mensis Februarij, post anatomen:
D. MATHEUS LEPI, Britanicus, D. Stephani, lauream in Phil. et Med. consecutus est.

111. 1683, Indictione VI, die Martis 3 mensis Augusti, hora 13 in c.:
D. JOANNES TOWNELEIJ, Anglus, D. Richardi filius, lauream in Phil. et Med. cons. est.

112. 1683. Indictione VI, die Mercurij, 22 mensis Septembris, hora 14 in circa:
D. ODOARDUS ROOPER, Anglus, Londinensis, D. Gulielmi filius, lauream in Medicina tantum, consecutus est, quia antea in Almo Gymnasio Lovanij, Doctor in Philosophia approbatus fuerat. Fidem fecerunt. EE.DD. Rev. Pater. Patritius Greems, Hibernus, D. Bernardi filius, *Ord. August.* et Doctor Joannes Towneleij, Anglus, D. Richardi filius.

113. 1684, Indictione VII, die Martis 28 mensis Martij, hora 14 in circa:
D. THOMAS WALKELEY (WAKELY), Sommersettensis, Anglus, D. Joannis filius, lauream in Phil. et Med. consecutus est, et postea:

114. D. RICHARDUS BLACKMORE, Anglus, ex Agro Wiltoniensi, q.m. D. Roberti filius, *Magister Artium*, lauream in Medicina tantum, consecutus est, quia in Almo Gymnasio Oxoniae, Doctor Philosophiae die 3 mens. Julij A. 1676, nominatus fuerat.

115. 1684, Indictione VII, die Mercurij 13 mensis Decembris, post lectiones, hora 18:
D. NATHANIAEL SPEYE, Anglus, ex Comitate Cornubiensi (Cornwall), D. Gulielmi filius, lauream in Phil. et Med. consecutus est, et postea:

116. D. CORNELIUS CALLOW, Anglus, Londinensis, D. Roberti filius, lauream in Medicina tantum, consecutus est, quia privilegium suum Philosophiae presentavit datum Cantambrigiae die 11 mensis Julij A. 1684.

117. 1685, Indictione VIII, die Martis 2 mensis Octobris, hora 14 inc.:
D. NATHAN LAEY (alias LACIJ, LACY), Anglus, Londinensis, D. Nathan fil. lauream in Phil. et Med. consecutus est.

118. 1686, Indictione IX, die Veneris 21 mensis Junij; hora 12:
D. DANIEL O'KEARNY, Hibernus, q.m. Gulielmi filius, lauream in Philosophia et Medicina consecutus est.

119. 1688, Indictione XI, die Martis 13 mensis Julij, hora 13 inc.:
D. GILBERTUS HEATHCOTE, Anglus q.m. D. Georgij filius et:

120. D. JABEZUS CALJ (alias CALF), Anglus, q.m. D. Roberti filius, lauream in Phil. et Med. consecuti sunt.

121. 1688, Indictione XI, die Martis 20 mensis Julij hora 12 in c.:
D. THOMAS ABRAMUS, Anglus, D. Nathanaelis fil. et:

122. D. ROBERTUS SHEPPARD, Anglus, D. Roberti filius, lauream in Philosophia et Medicina consecuti sunt.

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123. 1691, Indictione XIV, die Veneris 16 mensis Februarij, hora 17 in c.:
D. ROBERTUS BILL, Scotus, D. Roberti filius, lauream in Philosophia et Medicina consecutus est.

124. 1693, Indictione I, die Martis 17 mensis Novembris, hora 17 in c.:
D. JOANNES BAPTISTA NIPHO, Anglus, Londinensis, lauream in Phil. et Med. consecutus est.

125. 1695, Indictione III, die Veneris 26 mensis Augusti, hora 13 in c.:
D. RICHARDUS MEAD, Anglus, Londinensis, lauream in Phil. et Med. consecutus est.

Anno

126. 1696, Indictione IV, die Jovis 12 mensis Januarij post lectiones de mane:
D. ROBERTUS LEIJ (LEY), Anglus, Londinensis, D. Thomae filius, lauream in Philosophia et Medicina consecutus est.

127. 1700, Indictione VIII, die Lunae 20 mensis Decembris, hora 17 in circa:
D. MARMADUCUS DRINKELL, Lancastrensis, Anglus, q.m. D. Thomae filius, lauream in Philosophia et Medicina consecutus est, et postea:

128. D. EDUARDUS SMITHSON, Anglus, Eboracensis, D. Thomae filius, lauream in Philosophia et Medicina consecutus est.

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129. 1706, Indictione XIV, die Sabbati 25 mensis Septembris:
D. JACOBUS TASBURGH, Anglus, D. Richardi filius, lauream in Phil. et Med. cons. est.

130. 1708, Indictione I, die Veneris 16 mensis Martij, hora 17 post lectiones:
D. JOANNES QUINTON, Anglus, D. Joannis filius, *more nobilium*, lauream in Philosophia et Medicina consecutus est.

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131. 1715, Indictione VIII, die Jovis 17 mensis Januarij, hora 17 post lectiones:
D. JOANNES ARNOLDUS (ARNOLD), Exoriensis Anglus, D. Gulielmi filius, lauream in Philosophia et Medicina consecutus est.

132. 1715, Indictione VIII, die Veneris 28 mensis Junij, hora 12 in circa:
D. HENRICUS LESLIE, Hibernus, Scotus, D. Caroli filius, lauream in Phil. et Med. cons. est.

133. 1717, Indictione X, die Jovis 17 mensis Julij, hora 13 in circa:
D. JOANNES MARSHALL, Edimburgensis, Scotus, D. Georgij filius, lauream in Medicina tantum, consecutus est, cum jam esset declaratus Philosophiae Magister in Atheneo Edimburgensi.

134. 1719, Indictione XII, die Martis 18 mensis Julij, hora 12 in circa:
D. JOANNES TIMPLITON (alias TEMPLETON), Hibernus, D. Joannis filius, lauream in Phil. et Med. consecutus est.

135. 1722, Indictione XV, die Sabbati, 21 mensis Novembris, hora 17 in circa:
D. JOANNES JNNES, Scotus, q.m. D. Joannis fil., lauream in Philosophia et Medicina consecutus est.

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136. 1732, Indictione X, die Jovis 26 mensis Junij, hora 12 in c.:
D. PETRUS VANTEYLINGER, Londoniensis, Anglicus, D. Joannis Londinensis filius, lauream in Philosophia et Medicina consecutus est.

137. 1735, Indictione XIII, die Jovis, 3 mensis Martij, hora 17 in circa:
D. JOANNES BEDFORD, Anglus, de Londino, D. Ill. mi Hilkiah (?) filius, lauream in Phil. et Med. consecutus est.

138. 1738, Indictione I, die Martis 15 mensis Julij, hora 12 in circa:
D. ROBERTUS WOOD, Hibernus, D. Alexandri filius, lauream in Philosophia et Medicina consecutus est.

139. 1739, Indictione II, die Jovis 16 mensis Aprilis:
D. GEORGIUS MAC-ANLAY (alias MACAULAY), Scotus, D. Archibaldi, lauream in Phil. et. Med. consecutus est.

140. 1744. Indictione VII., die Martis 13 mensis Octobris, hora 15 in circa:
D. GULIELMUS MORTON GLIKES (alias MORETON GILKES), D. Gulielmi filius Nobilis Angli, lauream in Philosophia et Medicina, *more nobilium*, consecutus est.

Anno

141. 1745, Indictione VIII. die Mercurij 5 mensis Majj, hora 13 in circa:
D. GEORGIUS GARNIER, qm. D. Pauli, Nobilis, Londinensis filius, lauream in Philosophia et Medicina, *more nobilium*, consecutus est.

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142. 1768, Die 3 Octobris:
D. GEORGIUS BLEY, D. Gulielmi, Anglici, filius, lauream in Philosophia et Medicina consecutus est.

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143. 1771, Die Mercurij 6 martij, hora 16 in circa:
D. GULIELMUS WYNNE, D. Riccardi Anglici filius, lauream in Philosophia et Medicina, *more nobilium*, consecutus est.

A Letter of Fothergill on Malignant Sore Throat, Human and Bovine (1747).

By R. W. INNES SMITH, M.D.

THROUGH the kindness of Mr. Charles Drury, the distinguished Sheffield antiquarian, I am able to bring before the Section a letter of John Fothergill's, addressed to Dr. Robert Key, of Leek. Dr. Key was a Quaker physician and practised for many years in Leek. He was born in 1708. He was entered at Leyden University, September 24, 1734, and graduated there September 3, 1736, with dissertation *De Hæmoptysi*. He died 1761. The letter is dated 1747, from London. Fothergill was then residing at White Hart Court in the City, and was evidently in full practice. He refers shortly to his early cases of the malignant sore throat. His celebrated pamphlet on this disease was not published till the following year. The most interesting part of the letter, however, is his reference to the cattle plague which had evidently broken out in Leek. His method of dealing with it is precisely the method of to-day.

A LETTER FROM Dr. JOHN FOTHERGILL TO Dr. ROBERT KEY, OF LEEK.

London, 29/7/1747.

DEAR FRIEND,

* * * * *

I have perused thy ingenious manuscript with pleasure and instruction. The substance of what the most judicious of the Ancients delivered on that subject is there laid together in a very pretty manner. And the rules with regard to the treatment of acute diseases are as just as they are necessary to be more attended to. As I have thy permission to keep it a little longer I'll give it a second perusal and return it thee by some safe conveyance.

We have lately had an uncommon kind of disorder appearing in some places. It can scarcely be called epidemick as it has lived through several seasons and only seems to affect here and there a family. Children are more especially exposed to it, and I have heard of instances where four out of five have been carried off by it in a few days. It is the Morbus Strangulatorius which appeared in Italy Sicily and Spain about a century ago and is described by several writers of that time. Aetius Cletus wrote a tract upon it. Cortesius in his miscellanys likewise describes it, also Severinus and many others. I have attended five within these few weeks who all recovered but one—a girl about 13 who died after about 19 hours illness. If thou hast none of the writers by thee who describe it, I'll give thee an account of what I observed in my next.

With regard to the disease amongst cattle I hardly know what to say. Prevention is the only certain cure, and if the magistrates are disposed to act for the good of their country, I apprehend they are sufficiently authorised. No fresh cattle must be brought into uninfected places on any pretence whatsoever: as strict a guard should be kept as its possible. The cheapness of cattle in infected places tempts some to run all risks and those who do offend should be made examples of. As I believe thou hast frequent access to persons of some influence, thou wilt be doing the country an important piece of service to prompt them to be vigorous. I know not whether it is even prudent to suggest a remedy if one knew it, which I confess I do not. Since by making tryals with these we keep the beast alive till the infection is maturated and tho' the cow recovers yet she has added something to the infectious taint capable of affecting the rest of the herd. Besides, there is this one circumstance which is enough to deter one from thinking that any medicines can be in general successful. Ruminant animals when in health take in what food they can; if they are attacked with any disease rumination ceases; if the quantity lodged in the paunch is great it corrupts and adds to the disease. Thus in the present case the cattle in a few hours pass from a very healthy to a very morbid state: they are seized with a rigor, a violent fever ensues; it produces all the effects upon the blood etc. as it would do in any other animal. If bleeding or other methods take off these first symptoms or mitigate them, yet, as another cause is every moment coming into play, I mean the putrescence from the corrupting food, the chance against their recovery is very great. About the third fourth or fifth day a scousing seizes them, sometimes sooner sometimes later, and the matter discharged is so acrid as to cause motions like as in a Tenesmus and often brings off blood. Bleeding at first with Nitre and absorbents plentifully, chalk for instance or powder'd oyster shells seem to be proper; the first attenuates; the last sheaths the acid of the grass in their stomachs and prevents the corruption from being so quick. When they begin to scowr, bole, warm aromaticks mineral acids wrapt up with mucilaginous substances would probably succeed best. But prevention is chiefly to be aim'd at. If a town or village agree amongst themselves not to buy in any fresh stock, at least from the southward or infected places; nor to have any intercourse if possible with such places, they may possibly escape. If any beast is seized kill it immediately and perhaps the infection may die with it.

* * * * *

Thy affectionate Frd

J. FOTHERGILL.

The three books referred to in the letter all find a place in Lettsom's edition of Fothergill's works, Vol. I, pages 345 to 347. Johannes Baptista Cortesius mentions the disease in *Miscellanea Medica*, 1625. Aetius Cletus, of Signia, in Italy, mentions it in his *De Morbo strangulatorio*, Rome, 1636, and Marcus Aurelius Severinus wrote a dissertation on it under the title of *Paedanchone Loimodes, seu de pestilente ac praefocante Pueros Abscessu*, 1643.

Section of Laryngology.

President—Dr. ANDREW WYLIE.

The Pathology of Œsophagectasia (Dilatation of the Œsophagus without Anatomic Stenosis at the Cardiac Orifice).

DEMONSTRATION OF FIVE FURTHER SPECIMENS.

By IRWIN MOORE, M.B.

THE following specimens of œsophagectasia, three of which I am able to demonstrate to-day through the courtesy of Mr. Grey Turner, Mr. Rutherford Morison, and Mr. Hadfield, are supplemental to the series which I published in 1919[1]. The two last were exhibited, as specimens of interest to the endoscopist, in the Museum of Oto-Rhino-Laryngology, British Medical Association Meeting at Bath, in 1924.

In addition to these three a further specimen from the Museum of the Royal College of Surgeons will be demonstrated, also another recently added to the same museum by Mr. A. L. Abel.

The investigation of Mr. Rutherford Morison's S-shaped specimen completes the opportunity of studying the three varieties or degrees of œsophagectasia which have, from time to time, been met with and described under the title of cardiospasm.

Through the courtesy of Mr. Grey Turner, I am also able to show you a specimen demonstrating a trans-thoracic anastomosis between the dilated œsophagus and the fundus of the stomach in a case of non-malignant stricture at the lower end of the œsophagus.

DILATATION OF THE ŒSOPHAGUS.

Specimen No. O.C. 2290A, in the Museum of the Royal College of Surgeons, exhibited by Dr. H. Handford,[2] of Nottingham, at a meeting of the Pathological Society of London, on October 18, 1886. It is an example of a very *moderate degree of fusiform dilatation*, with the *thickness of the wall only slightly greater than normal.*

Description.—An œsophagus, one half of which has been removed by a vertical incision, with the cardiac end of the stomach. It is 10 in. in length, and in the recent state and unopened it measured slightly over 2 in. in width at about its middle. Below this level it gradually narrows, and at 1 in. above the cardia has a diameter of $\frac{3}{4}$ in., and retains this width down to the cardia. The mucous membrane is smooth, and exhibits no ulceration, and there are no cicatrices or external adhesions at the lower extremity.

Microscopic examination shows that the thickness of the œsophageal wall is only slightly greater than normal. The mucous and submucous coats are *indurated and infiltrated with leucocytes, showing a chronic catarrhal change.* There is *no evidence of degeneration in the muscular coat.*

The specimen was taken, post mortem, from a man, aged 52, who died from aortic incompetence. He had suffered from regurgitation of food, occurring several times daily, for at least five years. The regurgitation was at times immediate, but retention for an hour was not infrequent. The heart was enormously dilated, also the thoracic aorta.

It is interesting to note that the œsophagus contained, post mortem, several ounces of fluid undigested food, chiefly milk and brandy.

[1] *Proc. Roy. Soc. Med.*, 1919, xii (Sect. Laryng.), 67.

[2] *Trans. Path. Soc.*, London, 1888, xxxix, 103.

32 Irwin Moore: *The Pathology of Œsophagectasia*

Handford says "there was no apparent cause for the obstruction of the œsophagus at its passage through the diaphragm, and the consequent dilatation above, unless, as seems probable, the very dilated aorta had caused some compression." He thinks it "quite possible that the dilated aorta may have to some degree pressed the œsophagus against the unyielding central tendon of the diaphragm, and so interfered with the free passage of food."

We now know that this was a case of cardiospasm.

Dilatation of Œsophagus the Result of Cardiospasm.

Specimen No. C. 4474.—Mr. Grey Turner's case from the University of Durham College of Medicine.

The œsophagus is 9 in. in length. It exhibits a *slight degree of dilatation throughout*. At the upper end the diameter of the tube is 1¼ in.; this is maintained for 2½ in., when a slight increase in calibre occurs, the measurement being 1½ in. in the following 2 in. A slight further increase occurs (1¾ in.) between this point and the diaphragmatic opening, where the tube becomes narrowed to 1 in., retaining that diameter down to the cardia. The areolar tissue immediately surrounding the œsophagus at the level of the portion of diaphragm preserved, shows no evidence of induration or constriction, and so far as can be seen there is no ulceration or cicatrization of the œsophagus in this situation, or of the part of the stomach immediately adjacent. *The wall of the œsophagus* is throughout *thinner than normal*, apparently from muscular atrophy, and the longitudinal folds are obliterated.

The stomach is dilated, its walls are thin and atrophied, and there is a gastrostomy opening near the pyloric end.

The specimen was removed post mortem from a woman, aged 58, who had suffered for twenty-three years from difficulty in swallowing and regurgitation of food. A mercury bougie always passed into the stomach with ease. Gastrostomy was performed, but the patient's state never improved and she gradually died. A subsequent examination proved the death to be due to generalized tuberculosis.

No microscopic examination has yet been made.

Achalasia of the Cardia.

(Idiopathic Dilatation and Hypertrophy of the Œsophagus.)

From the Pathological Department, General Hospital, Bristol.

I am indebted to Mr. T. W. P. Lawrence, F.R.C.S., for the following description of the specimen and microscopical report:—

The lower 6½ in. of an œsophagus, with the cardiac end of the stomach. The œsophagus has been laid open longitudinally, and has probably contracted somewhat in the fluid. The upper 5 in. is of about equal calibre throughout, about 2¼ in. transversely, with the tube laid out flat. Below this there is a *slight but distinct fusiform enlargement*, 1¾ in. longitudinally, and 2½ in. transversely at the middle, which narrows inferiorly to 1 in. in width, where it passes into the subdiaphragmatic portion of the tube, which maintains that diameter down to the cardia. The mucous membrane is normal throughout, except at the lower end where there is some slight superficial erosion; and it is freely movable over the subjacent muscle. There are no ulcers or cicatrices at the lower end of the œsophagus or in the adjacent portion of the stomach. *The muscle is hypertrophic*, except in about the lowest half-inch of the tube; at the upper part of the specimen it is $\frac{1}{10}$ in. in thickness, at the middle $\frac{2}{10}$ in., gradually thinning to $\frac{1}{10}$ in. in the lower dilated portion. There is no localized *hypertrophy corresponding to a sphincter*.

The specimen was taken from a man, aged 60, who had suffered from dysphagia for seven years. He had little difficulty in swallowing fluids, but solids required an interval of from five to ten minutes, and an average mouthful was usually regurgitated. For twelve months he had been losing flesh, and had developed a cough with abundant and offensive sputum. A bismuth meal remained in the lower end of the œsophagus for twenty-five minutes. Death was due to suppuration, with a unilateral bronchiectasis.

Microscopical Report:—

(1) *From near Middle of Specimen.*—The epithelium is present, but in most places the superficial layers are shed; in a few spots there is some hypertrophy. The submucosa is increased in amount, i.e., thickened and fibrosed, and the muscularis mucosæ hyper-

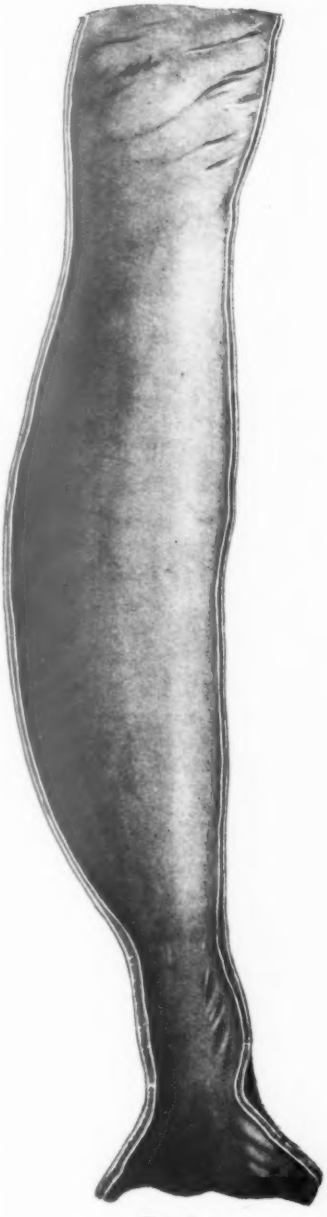

FIG. 1.

FIG. 2.

FIG. 1.—Fusiform dilatation of œsophagus. (Specimen No. O.C. 2290A in the Museum of the Royal College of Surgeons.) Mr. H. Handford's case.

FIG. 2.—Dilatation of œsophagus the result of cardiospasm. (Specimen No. C. 4474 in the Museum of the University of Durham College of Medicine.) Mr. Grey Turner's case.

34 Irwin Moore: *The Pathology of Œsophagectasia*

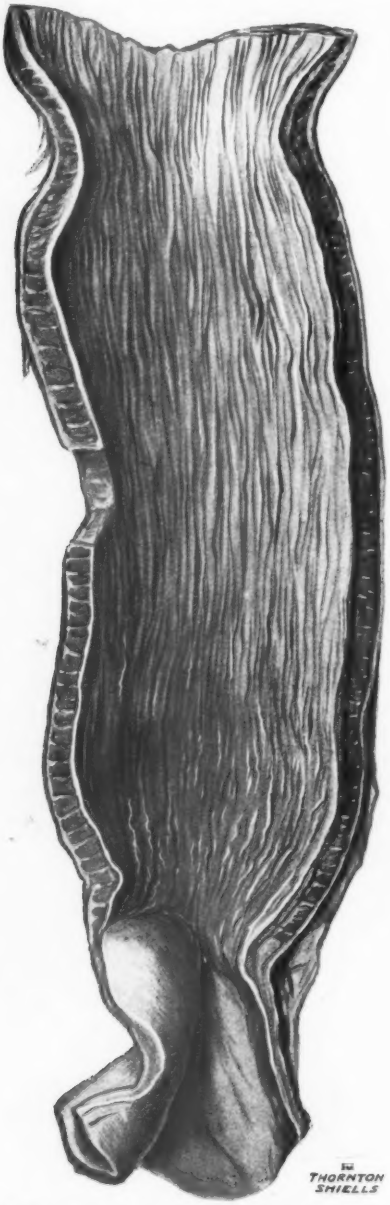

FIG. 3.—Idiopathic dilatation and hypertrophy of the œsophagus. (From the Pathological Department, General Hospital, Bristol.)

trophied. The circular layer of muscle is much hypertrophied, but there is no increase of fibrous tissue between the bundles. The same remark applies to the longitudinal layer. In both layers there is marked *hyaline degeneration of the fibres*, and where the bundles are cut transversely there are numerous areas in which *vacuolation of the fibres* is present, and many of the bundles appear to have undergone reduction in size (*atrophy*).

(2) *From Lower End of Œsophagus.*—The fibrosis is similar to that described. The epithelium is completely desquamated over considerable lengths. The hypertrophied muscularis mucosa has undergone *marked atrophy*, and has almost entirely disappeared in some spots. *Vacuolation and hyaline change* in the muscular layers are less marked (the nuclei stain better), but *atrophy of the muscular bundles* is more advanced.

ŒSOPHAGUS: FUSIFORM DILATATION.

No. O.C. 2299A, in the Museum of the Royal College of Surgeons. Presented by Mr. A. L. Abel. (Jacksonian Prize Essay, 1924.)

The lower part of the œsophagus, opened out. It is *greatly dilated*, measuring 5½ in. in circumference in the greater part of its extent, but at about 2 in. from its lower end it suddenly contracts and at the cardia has a circumference of only 1 in. Except in its lowest 1½ in. *the wall of the gullet is abnormally thick*, from hypertrophy of the muscular coat and chronic inflammation of the mucous membrane. In the lower 1½ in. the wall is thin. At about 4 in. above the lower end there is a localized bulging of the anterior wall, about 3 in. in diameter. Almost the entire surface of the mucous membrane is riddled with small, shallow, acute ulcers. There is no cicatricial contraction at the cardia, the connective tissue around the lower part of the gullet is normal.

From a man, aged 32, who complained of wasting and indigestion, which commenced at the age of 14, and was associated with vomiting immediately or within two or three hours after taking food. Ten months before admission to hospital the patient had a severe hæmatemesis, losing a large quantity of blood during four days. He became extremely emaciated and died with symptoms of broncho-pneumonia.

ŒSOPHAGUS: DILATATION.

Mr. Rutherford Morison's Specimen of S-shaped Œsophagectasia, from the Newcastle Museum.

(Description by Mr. T. W. P. Lawrence, Royal College of Surgeons.) A portion of the wall throughout its entire length has been recently removed, at my suggestion, in order to show the varied thickness of the wall.

The œsophagus is *grossly dilated in its entire length*, except its diaphragmatic portion and upper extremity, and forms a large S-shaped bend. At its upper end the œsophagus dilates rapidly, and at about its middle reaches a diameter of 3 in., below which it gradually diminishes in width, attaining the normal at the level of the diaphragm. The dilated portion forms a marked curve, with the convexity to the right. Excepting in the subdiaphragmatic portion and at the upper extremity, the muscular coat of the œsophagus is hypertrophied, but is *thinned out* in the part forming the convexity of the curve. The hypertrophy is most distinctly marked below the level of the diaphragmatic opening, in the position of the sphincter. No cicatrices or ulcers are present in the lower end of the œsophagus or adjacent part of the stomach. The lining of the œsophagus is smooth and without any definitely localized ulceration. The sac was at least three times the size of the stomach. A thick finger easily passed through the cardia. The stomach is small and atrophic.

Microscopic examination shows that the muscular coat in the region of the sphincter is hypertrophied, the epithelium in great part desquamated, and the *submucosa thickened and fibrosed*, and in many places densely *infiltrated with leucocytes*. The muscular fibres are normal. *Section of the thinner parts* of the œsophageal wall show still more extensive denudation of epithelium, with similar *fibrosis* and leucocyte infiltration of the submucosa. The muscular layer is also fibrosed in places and its fibres exhibit well-marked *hyaline degeneration* and *transverse fragmentation*, especially in the superficial layers.

From a man, aged 44, who had difficulty in swallowing from the age of 16. He was first seen in 1907, at 35 years of age—nine years before his death. No instrument could be passed into the stomach from above. The stomach was opened and the cardiac orifice

36 Irwin Moore: *The Pathology of Œsophagectasia*

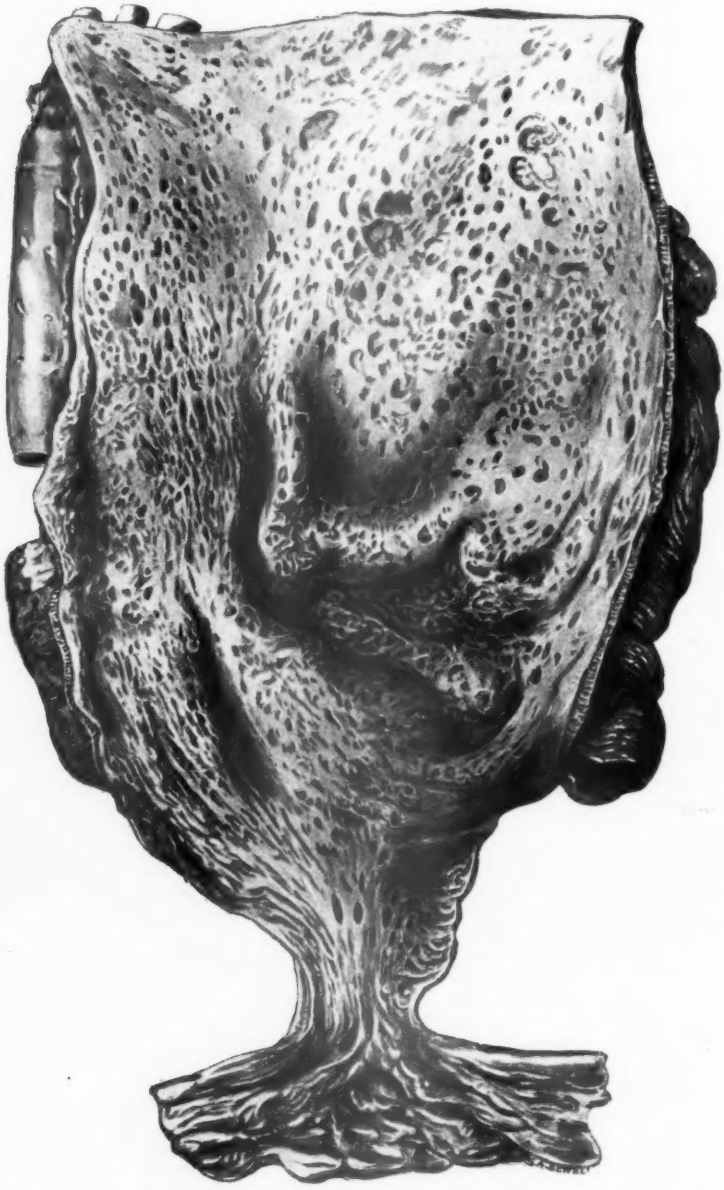

FIG. 4.—Fusiform dilatation of œsophagus. (Specimen No. 2299A in the Museum of the Royal College of Surgeons.) Mr. A. Lawrence Abel's case.

FIG. 5.—Idiopathic dilatation and hypertrophy of the œsophagus. (From the Museum of the University of Durham College of Medicine, Newcastle-upon-Tyne.) Mr. Rutherford Morison's case. In the preservation of this specimen in its receptacle the stomach has been twisted round; it ought properly to be directed to the left of the specimen.

38 Irwin Moore: *The Pathology of Œsophagectasia*

digitally dilated from below, with temporary relief of symptoms. Ultimately, however, the patient had great difficulty in swallowing, even fluids passing very slowly into the stomach; he became extremely weak and emaciated and died from starvation. There were no signs of disease in any other organ.

THREE VARIETIES OR DEGREES OF DILATATION.

(1) Fusiform, most frequently met with.
(2) Flask or pear-shaped, next in frequency.
(3) S-shaped, rarest of all.

(1) In the *fusiform variety*, which is most frequently met with, the cardia is the most dependent portion. According to Lambert there is no increase in length of the œsophagus. Walton is of the opinion that there is little if any increase, while Brown Kelly considers that lengthening is always present. It is probable, however, that lengthening only occurs in the S-shaped variety.

In the fusiform type the lumen of the œsophagus is increased from above downwards to a point approximately midway between its upper end—at the cricoid level—and its lower end, the cardia, and it then gradually diminishes in diameter until it passes through the diaphragm or enters the stomach.

Specimens showing various degrees of fusiform dilatation are:—

(*a*) St. George's Museum.[1] Showing *marked fusiform dilatation* involving the whole of the œsophagus from the cricoid to the cardia.

(*b*) No. 912A, St. Thomas's Hospital Museum[2] (measuring 2 in. in diameter at its widest part).

(*c*) No. 552, Guy's Hospital Museum[3] (measuring 6½ in. in external circumference at its widest part).

(*d*) No. 1522, University College Hospital Museum.[4]

(*e*) No. O.C. 2290A, Royal College of Surgeons Museum (Handford's case).

(*f*) Royal College of Surgeons Museum (Abel's case)—greatly dilated = 5½ in. circumference in greater part of its extent.

(2) In the *flask or pear-shaped variety* the lower third or two-thirds of the œsophagus is dilated, the lumen attains its greatest degree of dilatation just before its passage through the diaphragm—the dilated portion bulging over the surface of the diaphragm, and forming a kind of sulcus around the opening through the diaphragm, which is somewhat raised.

In this case also the cardia is the lowest and most dependent portion of the œsophagus.

Both of these forms are relieved by dilatation of the cardia. Illustrated by Specimen No. 1833, St. Bartholomew's Hospital[5] (measuring nearly 6 in. in circumference in its lower half).

(3) The *S-shaped variety* is the rarest form, and arises as follows: The upper end of the œsophagus opposite the cricoid cartilage, and its lower end at the cardia, being more or less firmly fixed points, the canal bends in those cases in which, as the dilatation of the œsophagus advances, there occurs also an *increase in its length*. This usually results in a curve towards the right and the dilated œsophagus rests on the diaphragm to the right and posterior to the cardia; from here it passes upwards, forwards and to the left, and enters the abdomen through the diaphragm at a high level. There is thus developed a reservoir or dead space, the most dependent portion of the œsophagus being below the level of the cardia.

The X-ray appearances of the S-shaped dilatation of the œsophagus are well

1 *Proc. Roy. Soc. Med.*, 1919, xii (Sect. Laryng.), 79, fig. 1.
2 *Op. cit.*, 83, fig. 5.
3 *Op. cit.*, 84, fig. 6.
4 *Op. cit.*, 87, fig. 9.
5 *Op. Cit.*, 86, fig. 8.

demonstrated in cases recorded by Lambert[1] and Walton.[2] The illustrations show, in antero-posterior views, the bend or S-shaped curve of the lower portion of the dilated œsophagus, while in lateral views they show the reservoir or dead space which lies on a lower level than the dome of the diaphragm where the œsophagus enters the abdomen.

Hypertrophy of the Muscular Coat.

Secondary or compensatory in character. Varies in degree in different cases, either in the same specimen or at different levels of the dilatation. Walton says that all cases show this change. Grey Turner's and Handford's cases, however, are exceptions (Irwin Moore).

The Bristol Hospital specimen shows *slight but distinct* fusiform dilatation with *marked hypertrophy* of the circular fibres the entire length of the œsophagus, except in about the lowest half-inch of the tube. There is no localized hypertrophy corresponding to the sphincter. The London Hospital specimen, No. H. 49,[3] shows the *hypertrophy* of the circular fibres lying almost entirely above the actual constriction, and in the wall of the lowest part of the dilated œsophagus, varying from 3 to 4·5 mm. in thickness.

The University College Hospital specimen, No. 4004,[4] shows *marked* fusiform dilatation with well-defined and pronounced hypertrophy of the circular fibres around the terminal portion of the œsophagus, i.e., the phreno-cardiac segment = cardiac canal. Very rare.

Walton, in 1925, in his paper on the "Surgical Treatment of Cardiospasm," attempted to define this disease as follows:—

"A condition of dilatation and hypertrophy of the œsophagus in which, on post-mortem examination, no obstruction can be found distal to the dilatation."

In view, however, of Grey Turner's recent case, in which there is dilatation with muscular atrophy in place of hypertrophy, also the specimen No. 550 in Guy's Hospital Museum, which I demonstrated in 1919, in which muscular hypertrophy is present at and above the cardia for a distance of 2 in., without any dilatation, it cannot be said that this definition covers the condition.

Shattock, in 1919, remarked that

"there is no specimen in which the muscular coat is atrophic, i.e., absolutely thinner than the wall of the normal œsophagus, but," he added, "an atrophic condition might be merely a secondary phenomenon and is not to be ignored."

Brown Kelly, in 1920, said:—

"It is significant that thinning of the wall has not been observed in œsophagectasia. On the contrary, in many specimens the thickness of the wall, i.e., the degree of muscular hypertrophy, corresponds to the amount of the dilatation, instead of the reverse, as might be expected. Thus, in spindle-shaped dilatations the wall often attains its maximum thickness where the gullet is widest, and in a lateral bulging the musculature has been found thicker than elsewhere at the same level. This relation between the thickness of the wall and the amount of dilatation, although holding good generally, is not invariable."

Walton also states that the more marked the dilatation the greater the hypertrophy. The specimen, however, in the General Hospital, Bristol, is an exception to this rule.

I am able to demonstrate to-day two cases of atrophy of the wall:—

(1) In Grey Turner's specimen the wall of the œsophagus throughout is seen to be thinner than normal, apparently from muscular atrophy, and is confirmed microscopically.

1 *Surgery, Gynæcology and Obstetrics*, 1914, xviii, 1-9.
2 *Brit. Journ. Surg.*, 1925, xii, 724, fig. 442; 726, fig. 445; 729, fig. 451.
3 *Proc. Roy. Soc. Med.*, 1919, xii (Sect. Laryng.), 90, fig. 12.
4 *Op. cit.*, 88, fig. 10.

40 Irwin Moore: *The Pathology of Œsophagectasia*

(2) In Rutherford Morison's specimen there is only a local thinning out, viz , in that portion of the wall forming the convexity of the S-shaped curve; this is confirmed by the microscope.

It may be remembered that in 1919, in connexion with the series of specimens I then collected together, I drew attention to the interesting fact that in a certain number of cases it is recorded that the contents of the dilated œsophagus remained *in situ* after death, and that all active muscular contraction having ceased, the obstruction at the lower end could only be attributed to the mechanical pressure of the surrounding parts, as in the retention of urine, e.g., in the bladder after death.

Walton says that

"it is of interest to note that such cases showing hypertrophy will usually hold fluid after death, as with the stomach with congenital pyloric stenosis, so that the condition is not one of pure spasm."

Fluid contents were found, post mortem, in the following specimens :—

(1) Handford's case. Fusiform dilatation with only very slight hypertrophy.
(2) St. George's Hospital. The œsophagus when opened contained fully a pint of milk. In this case there was great fusiform dilatation and muscular hypertrophy involving the whole of œsophagus to stomach.
(3) St. George's Hospital, No. 21A. Moderate dilatation and hypertrophy; contained liquid food.
(4) St. George's Hospital, No. 21B. Dilatation and muscle much hypertrophied; contained much modified food.
(5) University College Hospital, No. 1522. Fusiform dilatation and slight hypertrophy extending to the gastric orifice; filled with undigested food.
(6) London Hospital. Dilatation and hypertrophy; contained about 10 oz. of grey-brown fluid.
(7) Brown Kelly's second case of dilatation and hypertrophy, recorded in 1920. On removal the œsophagus was filled with water and none escaped by the lower end.

So that out of fifteen cases, six contained fluid contents after death, and one held water on being filled after death.

ŒSOPHAGO-GASTROSTOMY.

(Mr. Grey Turner's case.)

The specimen shows a trans-thoracic anastomosis made between the dilated œsophagus and the fundus of the stomach in a case of intractable non-malignant stricture at the lower end of the œsophagus.

The lower 5½ in. of an œsophagus, with the cardiac end of the stomach and parts of the adherent diaphragm and left lung. The last 2½ in. of the œsophagus has a funnel shape, rapidly narrowing down to the cardia, where it is tightly stenosed and only admits a probe. The upper 3 in. of the œsophagus preserved exhibits a *moderate cylindrical dilatation*, its diameter being 1¼ in., and the *muscular coat is hypertrophied* right down to the stricture. Instead of passing straight downwards into the stomach, the œsophagus is bent at its lower part, the last 2½ in. occupying a more horizontal position, with the constricted lower extremity directed to the right. In this situation an oval aperture of communication, measuring about ½ in. in its chief diameter, has been made between the posterior wall of the œsophagus and the fundus of the stomach, at a distance of 2½ in. from the cardia. A portion of the fundus of the stomach is seen above the diaphragm—seen in section of the left side of the œsophagus.

The patient, a male, aged 19, had suffered for years from the stricture, which resisted all methods of treatment. The operation was carried out with considerable difficulty, and though the patient's convalescence was somewhat stormy, he ultimately made an excellent

recovery and was delighted with the result. He unfortunately died, however, two years and eight months later, as the result of being knocked down by a motor car.

The original and strictured opening between the œsophagus and stomach can be seen.

Grey Turner has recorded a second case, in which he performed a trans-peritoneal anastomosis between the dilated œsophagus and the stomach within the abdominal cavity. The œsophagus was first loosened from the diaphragm and was drawn into the abdomen. This loosening process was fairly easily accomplished. He made a direct anastomosis between the portion of the œsophagus which had been drawn into the abdomen and the fundus of the stomach. In this case the result was immediately successful. The patient had no trouble during convalescence, and remained perfectly well when Mr. Grey Turner saw her more than a year after the operation.

Microscopical Note and Summary.

The fibrotic and degenerative changes in the submucous and muscular layers in two of the specimens of œsophagectasia which I have demonstrated, confirm the microscopical findings of Dr. John Anderson, Pathologist to the Victoria Infirmary, Glasgow, in the three cases of œsophagectasia recorded by Dr. Brown Kelly in the Semon Lecture.[1]

It is highly probable that, as suggested by Brown Kelly, destruction of the nerve mechanism of Auerbach's plexus, owing to chronic fibrosis and degenerative changes, may account for the loss of the normal muscular contraction and relaxation, and result in a greater tendency to abnormal reflex overaction of the circular fibres.

But it has not been possible at present to ascertain what is the cause of these degenerative changes. It seems likely that some toxic condition may eventually be found to be the causative factor.

Mr. Granville Hey said he was not a member of the Section, but had been drawn to this meeting to hear this contribution. A few years ago he had had a patient—still living—whose case was an example of the variety last described. The patient complained of chronic vomiting; the food vomited had evidently been ingested three or four days previously, yet no change beyond maceration had taken place. He concluded that a large pressure pouch was present and as the vomiting had not been relieved by any other means he had performed gastrostomy by Senn's method. The vomiting had then ceased because the patient fed herself through the gastrostomy tube; she consequently put on flesh, and was quite comfortable for several years. Last year she returned to consult him, as vomiting had recommenced. He had then given her a barium meal, and under X-ray examination the barium was seen to go, not down into the stomach but to the base of the right chest cavity, and to lie over the dome of the diaphragm. Skiagrams of the chest showed a large triangular opacity entirely to the right of the spinal column. Various means were taken with a view to stopping the vomiting, but these proved unavailing. Finding that the pouch was so low down and not being able to explain the facts, as he was at that time ignorant of such a condition, he opened the chest through the right tenth intercostal space, found the sac without any great difficulty, and brought it the surface, suturing its covering to the parietal pleura, opening it, and suturing its mucous lining to the skin. Healing took place quickly and aseptically. This procedure had drained the pouch, and had given much relief, as the vomiting again ceased and the patient put on flesh. The patient still fed herself through the gastrostomy tube, but occasionally some of the food came out through the pleural opening. He (the speaker) concluded that this was an example of the sigmoid variety of sac. The condition in this case had never been supposed to be due to anything other than a pressure pouch.

An attempt was being made to dilate the cardia and if this was successful then the fistula in the chest wall would be closed.

[1] The Semon Lecture, University of London, delivered in the Hall of the Royal Society of Medicine, December 2, 1926, *Journ. Laryng. and Otol.*, 1927, xlii, 221.

CASES AND SPECIMENS.

Congenital Bilateral Occlusion of the Posterior Choanæ.

By HERBERT TILLEY, F.R.C.S.

G. R., AGED 5, female. The complete nasal obstruction noticed at birth caused much trouble for a week owing to "attacks of suffocation." Discharge from each ear when eight or nine months old; this lasted more than two months and then ceased without causing any deafness.

October 10, 1926.—Complete absence of nasal respiration: nasal cavities filled with mucus. Each posterior naris was completely occluded by a diaphragm which had a membranous centre and bony circumference. The former was perforated by firm pressure with a blunt probe.

October 14.—Under general anæsthesia, the membranous portions of each diaphragm and also a considerable mass of adenoids were removed by sphenoidal punch forceps.

For a week afterwards fair nasal respiration was possible, but gradually the obstruction increased in spite of daily applications of silver nitrate, 1 dr. to 1 oz. Therefore, on December 9 I removed the posterior edge of the vomer with a small chisel and forceps, so as to cut away the inner circumferences of the bony portion of the diaphragms. Instrumentation was guided by the left forefinger in the naso-pharynx. The child has now perfectly free nasal respiration and sleeps with closed mouth.

There is a general lack in the development of this patient which is in striking contrast with that of her sister, who is a year older.

Discussion.—Mr. A. J. WRIGHT said that at the Glasgow Meeting of the British Medical Association, he had read a short paper dealing with this condition in two sisters who had reached the ages of 10 and 13 respectively when the operation was performed. The point he had emphasized was the extremely good general development of the patients.

Mr. T. B. LAYTON said that apparently this child had never breathed through her nose, yet the arch of her palate was perfectly developed. This was one of the many pieces of evidence that the so-called typical "adenoid facies" had nothing to do with adenoids.

Mr. TILLEY said that at the first operation only the membranous portions of the diaphragms were broken down. The patient progressed very well for several days, but granulations formed and obstruction recurred. At the second operation the posterior margin of the vomer was removed as indicated. The condition was a rare one in a child. The fact that the child had not previously breathed through her nose might partially account for the lack of development as well as for the apparently senile appearance.

Growth on Palate: for Diagnosis and Treatment.

By H. BELL TAWSE, F.R.C.S.

E. A., FEMALE, aged 35. Ten years ago she noticed a fleshy-looking lump in the roof of the mouth. It caused her no trouble then, nor has it done so since. She is anxious to have a denture fitted, but the dentist refuses to make one until I give permission. The mass feels firm and fibrous and has enlarged since I saw it two years ago. Just above it there is a slight defect of the hard palate, and both the hard and the soft palates in this locality have a nævoid appearance.

Suggestions are asked for as to diagnosis, treatment, and the wisdom of having a denture fitted.

Wassermann reaction, negative.

Discussion.—Mr. H. TILLEY suggested placing a small quantity of radium in the swelling. An attempt at removal of the growth would probably result in perforation of the palate, which would be a more troublesome condition than the present one. But before adopting any treatment the speaker advised the exhibitor to read the monograph by the late Mr. Stephen Paget on "Tumours of the Soft Palate," describing adenomata of the palate as well as other new growths.

Mr. C. A. S. RIDOUT suggested that a portion of the growth should be removed for microscopical examination; ideas as to treatment could be formed more satisfactorily afterwards.

Mr. R. A. WORTHINGTON said that when he had removed the soft palate in adults, he had been surprised to find how little inconvenience had resulted from the procedure. There had been practically no interference with speech, and shortly after the operation there had been no trouble in swallowing. If the tumour were removed in this case and a denture fitted, he did not anticipate any inconvenience to the patient.

Sir WILLIAM MILLIGAN said that as this growth was not giving trouble it should be left alone, and he did not see why the patient should not have a denture; it could be so made as to encircle the growth. The growth might be a fibro-adenoma.

Dr. JOBSON HORNE observed that in the agenda Mr. Bell Tawse had asked for suggestions as to diagnosis and treatment. Extreme diffidence had been shown by members in offering these. That diffidence was not to be wondered at when one had read the discussion on another case of disease of the palate shown by Mr. Tawse on December 4, 1925.[1] The numerous suggestions then made as to diagnosis included chronic glanders, syphilis, foot and mouth disease, simple atrophic ulcer, tuberculosis, lupus and simple spreading ulceration. The Members were indebted to Mr. Tawse for showing the case at two subsequent meetings, and a final diagnosis of malignant disease had been arrived at. It was remarkable that in that discussion no mention had been made of malignant disease, especially so, since malignant disease was diagnosed more often than it existed.

With regard to the present case he (Dr. Jobson Horne) agreed that the growth was firm and fibrous and he was in favour of its removal.

Mr. BELL TAWSE (in reply) said that he had intentionally omitted to remove a piece for examination, as he wished to hear the diagnoses of Members. He would remove a piece—which he thought could be done without danger—and report the examination of it to the next meeting. His view was that it was a dermoid; there was much nævoid tissue in the vicinity. It might be a parotid tumour, and if cartilage were found inside it he would not feel surprised. Perforation was very likely to follow removal by operation, and in these cases his experience was that the fitting of a denture in such a way as to prevent food getting between the palate and the denture was almost impossible.

Styloid Process Projecting into the Left Tonsil: Skiagram shown.

By H. BELL TAWSE, F.R.C.S.

MRS. C., aged 42. Has septic tonsils, which at times are inflamed. On one occasion, having a sharp pain in the left tonsil, she put her finger in and felt a hard projection. A similar one could not be detected on the other side, so she examined the projection several times daily and soon concluded that it was growing larger and was probably a cancer.

The left styloid process appears to have been fractured. Was this an accident?

On the right side this process is longer than normal.

Discussion.—Mr. A. J. WRIGHT said he regarded this as a clinical entity. A styloid process projecting into the tonsil produced symptoms. He himself had operated in two such cases. One patient had refused operation for a similar condition in which the styloid process could be felt.

[1] *Proc. Roy. Soc. Med.*, xix, No. 4, February, 1926. See Laryng. pp. 23, 24, 25.

44 Guthrie: *Styloid Processes*: Wright: *Foreign Body in Bronchus*

Elongated Styloid Processes and the Stylo-hyoid Arch in Animals: Epidiascope Demonstration.

By DOUGLAS GUTHRIE, M.D.

DR. GUTHRIE said that in the majority of animals the hyoid bone was connected with the base of the skull by a chain of bones, and the styloid process, which was peculiar to man, represented the upper two ossicles, tympano-hyal and stylo-hyal, fused together and to the temporal bone. Occasionally a complete stylo-hyoid chain was found in man. The styloid process seldom exceeded 1½ in. in length, and a specimen longer than 3 in. (78 mm.) was a great rarity. It was difficult to estimate the frequency of those long styloids from museum specimens as the slender twig of bone was so frequently fractured in preparing the specimen. One could readily understand how such a fracture might occur during life from any force, such as the finger, applied to the tip of the process of the tonsil region. In two cases the speaker had encountered a long styloid process during tonsillectomy.

Foreign Body (Nut) in the Bronchus: Skiagrams of Chest Shown.

By A. J. WRIGHT, F.R.C.S.

A FEMALE child, aged 2 years, was admitted to the medical wards as a case of suspected laryngeal diphtheria. Some stridor was said to be present, but the patient was afebrile and the pharynx was normal. After a fortnight, a swinging temperature developed. When first seen by me, nearly four weeks after admission, the child was very ill, with much general toxæmia. An asthmatoid wheeze was present and the base of the right lung showed consolidation, with absence of breath sounds; the heart was displaced to the right side. The apex of the right lung, and the whole of the left lung, showed signs of emphysema. The diagnosis made was that of bronchial obstruction, probably by a vegetable foreign body. Bronchoscopy was performed with a 4 mm. tube, without an anæsthetic. The larynx was inflamed and, directly the tube entered the right main bronchus, several drachms of pus gushed out. As it was impossible to clear the bronchoscope, it was removed, and a fragment of nut was found impacted in its end. Recovery was uneventful.

The three skiagrams show the condition before the removal of the foreign body and two stages of resolution in the lung afterwards. The case typically illustrates an unrecognized organic foreign body in a bronchus, and also shows how rapid and complete resolution may be, even after five weeks of severe sepsis in the lung.

[I also show a skiagram of another case, which illustrates the fact that foreign bodies are not always discovered at first. The child in this case was brought to hospital with a very clear history of inhalation. She was sucking a pencil, when she took a deep breath and the pencil disappeared. The first skiagram taken had shown nothing, but the second disclosed the very faint lines indicated, due to the pencil being on edge. There was no particular difficulty in getting it out.]

Discussion.—Sir WILLIAM MILLIGAN said these cases were very dangerous; they were very apt to result in septic broncho-pneumonia. He (the speaker) had once removed a lentil bean from a bronchus in which it had been lodged for ten days. He thought he had removed the bean entirely, but the temperature did not fall, and death ultimately took place. The post-mortem examination disclosed a very small portion of the husk of the bean, which had become detached and which apparently had been responsible for the septic pneumonia. When the foreign body was soft and pultaceous, it was very important not to use forceps with sharp prongs for its extraction. The best kind was a sort of midwifery forceps on a very small scale, which enabled the foreign body to be grasped without causing laceration.

Mr. F. B. GILHESPY said that in taking a skiagram it was advisable to use lipiodol, which would show up the bronchial tree, also, perhaps, the foreign body.

Mr. M. VLASTO said that some years ago he had had a case almost identical with the one mentioned by Mr. Wright. A child had been admitted to hospital with physical signs of an inflammatory affection of the left lung. For many weeks there was a hectic temperature and a marked leucocytosis. No history could be obtained of aspiration of a foreign body, and radiography did not support such a diagnosis. He (the speaker) had examined the child by the bronchoscope; he was then extremely emaciated and almost moribund. When the distal end of the bronchoscope was introduced into the left bronchus a pultaceous mass was encountered which was coughed up through the lumen of the tube, together with a gush of fœtid pus. Dr. Sidney Owen, who was auscultating the chest, stated that he then heard breath sounds entering the left lung, and advised me to desist from further examination in view of the child's precarious condition.

Recovery was rapid and complete. It was remarkable to note the rapidity in the fall of the temperature, and in the degree of the leucocytosis.

Dr. IRWIN MOORE said that he had designed a pair of forceps, five years ago, of the kind Sir William Milligan advised for use in these cases.

A Laryngeal Case for Diagnosis.

By C. A. S. RIDOUT, M.S.

PATIENT, male, aged 59. History of hoarseness since August, 1926, followed by periods of improvement, but worse last few weeks. First seen February 9, 1927.

Laryngeal Appearance.—Right cord much thickened, movements limited, a definite prominence noticed over processus vocalis of right arytænoid. Below the anterior end of the left cord is a subglottic swelling, small but definite.

A small fragment, obtained with difficulty from the right cord, showed considerable keratinization of epithelium but no evidence of active changes.

Case of Nodular Laryngitis treated by Galvano-cautery.

By Sir JAMES DUNDAS-GRANT, K.B.E., F.R.C.S.

MRS. H., aged 42, mother of a large family, was seen in the Throat Department of the Cancer Hospital, in September, 1925, complaining of hoarseness and "a feeling of something in her throat." She was in great anxiety as to the possibility of this being cancer. I found a small œdematous swelling on the edge of the left vocal cord at the junction of the anterior and middle thirds. There was at the same time chronic rhinitis with pseudo-papillomatous swelling of the left inferior turbinated body. She attended hospital from time to time without any material diminution of the hoarseness until I applied a fine galvano-cautery point to the outgrowth, when the voice soon recovered the present considerable degree of clearness. The vocal cord is slightly thickened but perfectly smooth.

Epithelioma of Left Vocal Cord.

By L. GRAHAM BROWN, F.R.C.S.

C. W., MALE, aged 57, seen August 17, 1926. History of hoarseness for four months.

The growth was ulcerated, small, circumscribed and situated about the middle third of the left cord. The cords were freely movable.

46 Brown—Whale—Jewell—Vlasto

Operation, August 25, 1926.—Intratracheal ether, no preliminary tracheotomy tube used. A large window resection of the cartilage of the left thyroid ala was made, the growth and a large portion of the mucosa being removed. The wound was completely sutured at the close of the operation.

Pathological report: Epithelioma.

The patient made an uninterrupted recovery, could swallow fluids on the second day, his temperature was normal after the first twenty-six hours, the wound healed by first intention, and he left hospital a week later. Shown for the purpose of hearing opinions about the advisability of the routine use of a preliminary tracheotomy.

Case for Diagnosis.

By H. Lawson Whale, F.R.C.S.

F. C., aged 45. *History.*—Gunshot wound in mouth during the late war; he says this involved only the tongue. Denies syphilis; family healthy. For one year has had a swelling on left side of neck. Dysphagia and dysphonia two months.

Fauces.—Velum palati short. Uvula absent. Slough on lower pole of left tonsil. Right side, posterior pillar absent, or represented by a mass simulating upper pole of tonsil. Left side, posterior pillar hypertrophied, ending freely below in a pedunculated mass.

Neck.—Left submaxillary lymphatic gland palpable.

Larynx concealed by epiglottis which is all notched, red, fleshy and amorphous.

Wassermann result not yet available.

Ulceration of Left Vocal Cord.

By W. H. Jewell, M.D.

F. M., male, aged 48.

Huskiness of six months' duration; no cough; very slight expectoration; Wassermann negative. The right vocal cord is active and thickened and the epithelium is proliferated and macerated. The left vocal cord is ulcerated and at times there is some impairment of movement.

Laryngeal Case for Diagnosis.

By M. Vlasto, F.R.C.S.

Patient, male, aged 30, has occasionally had huskiness of the voice since the war. For the past year he has been definitely hoarse, and the hoarseness is increasing.

Wassermann negative; lungs reported clear. Examination of larynx showed both cords moving well; the right vocal cord was thickened irregularly; no definite ulceration was seen.

February 15, 1927.—A piece of the right vocal cord was removed by the direct method through Haslinger's directoscope. [Microscopic section shown.]

Discussion.

Mr. C. S. Ridout said that in his case he had removed a further fragment, which proved the growth to be epithelioma; he asked whether the operation indicated was laryngo-fissure or complete laryngectomy.

Sir StClair Thomson said that in Mr. Ridout's case the diagnosis was settled by the microscope, but even without that confirmation Members would have noticed the impaired

movement of the right cord. Yet the right cord, though thickened, was not very much involved. It was, however, sluggish in movement, and this afforded the valuable information that the growth was extended further than the surface of the cord. It did not extend to the attached border of the cord; it was rather in the subglottic region. As his (the speaker's) own figures showed, the prognosis of cases with the cord more or less fixed was not favourable. The growth extended across to the under surface of the opposite cord, though the cord itself was not invaded. The growth might spread round the anterior commissure to the opposite side without involving the surface of the opposite cord. He feared that in Mr. Ridout's case the condition was too far advanced for laryngo-fissure. The only chance of success would be afforded by complete laryngectomy.

With regard to Mr. Graham Brown's case, one was rather at a disadvantage because the exhibitor had not said what advantages he claimed for his method of dealing with these cases. The case certainly showed that the operation could be done from the side, and without a tracheotomy. But a few odd cases of this kind did not help to establish a principle. Malignant disease of the endolarynx had even been cured by removal through the mouth. He had himself had such a case, and Lynch, of New Orleans, had published records of nine cases, in five of which the patients had been alive without recurrence of the growth at periods of from two to five years afterwards.[1] Yet, no one who had had experience of laryngo-fissure would dream of recommending a patient to undergo a radical operation attempted through the mouth. With regard to so-called "window" resection: in that operation the first principle in the surgery of malignant disease was neglected, viz., that of free exposure, so that the surgeon could first see the full extent of the disease and plan his procedure accordingly. The "window" operation also deprived the surgeon of the valuable aid of touch. In the next room was a case which might be tuberculous, or might be malignant. In the laryngo-fissure operation the diagnosis might be settled by using the finger, since malignant disease of the larynx was firm to the touch, whereas tuberculous tissue was soft and gelatinous. He (the speaker) had made one mistake in such a case, and had published a report of it.

With regard to a tracheotomy: why should not that be performed? It was quite simple, and it fulfilled the great surgical principle, of doing everything for the safety of the patient.

Mr. Graham Brown's result was excellent but evidently he had not met with hæmorrhage which might occur unexpectedly during the operation, just at the end, or a few hours afterwards. The blood might pass downwards and do harm before a tracheotomy could be performed. Tucker, in Chevalier Jackson's Clinic, had carried out fifteen laryngo-fissures without tracheotomy; but in two he had to perform tracheotomy in haste, and it had also been necessary to plug the larynx, which was a very bad thing for it. One patient had died within a week, and in the other such diffuse granulation tissue had been produced by the operation that the tracheotomy-tube had to be put back two months afterwards. It was a method not to be encouraged. In laryngo-fissure a constant danger—and the only one—was the descent of blood. In the early cases operated upon forty years ago the patients had nearly all died because there had been no provision against this occurrence. Bleeding might occur when it was least expected, and if there was not a tracheotomy tube *in situ* the patient's life would be lost from this cause. Until every operator published all his statistics, it would not be known how many of such cases occurred. Since Sir Felix Semon's time, the only complete statistics on the matter which had been published in this country were those of Mr. J. S. Fraser, of Edinburgh, and his (the speaker's) own. He had placed records of all his results fully and freely at the disposal of his colleagues.

Mr. T. B. LAYTON drew attention to the fact that in two of these cases microscopical examination had been made of pieces removed and in neither case did it give any help.

Mr. R. A. WORTHINGTON said that after having done several laryngo-fissures, he had at one time believed that "window" resection was possibly a better method. Since he had come to this conclusion, however, two of his patients had been operated upon by the "window" resection method, and in both cases fistulæ had developed afterwards, an occurrence which had somewhat prejudiced him against the method.

Mr. H. TILLEY said he considered that in Mr. Jewell's and Mr. Vlasto's cases the growths were epitheliomata.

[1] *Trans. Amer. Laryng. Assoc.*, 1920, xliii, 119.

48 Howarth: *Œsophageal Speculum;* Graham Brown: *Clamp Forceps*

Sir WILLIAM MILLIGAN said that two important questions were: (1) Should "window" resection be done at all? (2) Should tracheotomy be done? The second question had been discussed fully by the Section a year or two ago, and the opinion of Members had been divided. His own view was that tracheotomy should always be done, as it was a great safeguard, even although it might not be actually necessary in every case.

"Window" resection departed from the ordinary canons of surgery, for if there was one thing desirable in dealing with malignant disease it was to expose the growth; he had never been satisfied with the view afforded by window resection, and if hæmorrhage occurred its control was very difficult.

Mr. GRAHAM BROWN (in reply) said that his reason for showing this case was that it was an isolated one of epithelioma, in which he had obtained a very careful and full view of the growth, and had determined that, as it was so small, it could be removed in a certain way. At the operation he had had an intratracheal anæsthetic administered very efficiently, the tube being passed well below the growth. He had had experience in "window" resection, and decided to proceed in that manner. He took a large piece of cartilage away, thinking it would be better to do that than carry out a laryngo-fissure. His operation practically meant the whole thyroid being divided, an arch being left to retain the framework of the larynx. There had been very little hæmorrhage; a small vessel had bled in the arytænoid region, but he was able to ligature it. As progress had seemed to be quite favourable, he had decided to close the wound entirely, with the result described.

Mr. HAROLD BARWELL said that the more he saw of these interesting lesions on the vocal cords the more diffident he felt about making a diagnosis; in Mr. Jewell's and Mr. Vlasto's cases it was difficult to make a "snap" diagnosis, when one had only a short time in which to examine. His own impression was that both were tuberculous. There was reason for some contrary suspicion in Mr. Jewell's case, but the opposite cord seemed to be considerably further involved than he (the speaker) would expect to find in the case of a new growth.

Mr. JEWELL (in reply) said he was inclined to accept Sir StClair Thomson's opinion; he would have a skiagram taken. He thought the case would probably prove to be malignant rather than tuberculous, considering the rapid destruction of the cord during the past three weeks without any constitutional disturbance, the sputum being negative for tuberculosis, and the fact that a physician had been unable to find any evidence of tuberculosis in the lungs.

Dr. JOBSON HORNE referring to Mr. Jewell's case agreed with Mr. Barwell, that it was not obviously one of malignant disease.

Mr. VLASTO (in reply) said that Dr. Elworthy had made a careful examination of the microscopical section in his (the speaker's) case and had reported the condition to be one of carcinoma. He (Mr. Vlasto) would therefore perform laryngo-fissure.

An Œsophageal Speculum with Distal and Proximal Illumination.

By WALTER HOWARTH, F.R.C.S.

THIS short tube has been found very useful in dealing with foreign bodies in the deep pharynx and upper end of the œsophagus, and also for the delimitation of growths in this region. The additional proximal illumination has been fitted to my other œsophagoscopes, which are also shown; it is simply an adaptation of the Hasslinger light.

A Pair of Hæmostatic Clamp Forceps used in Enucleation of Tonsils.

By L. GRAHAM BROWN, F.R.C.S.

THREE patients recently operated upon by the above method were exhibited to show the present condition of their faucial pillars and tonsillar fossæ.

Section of Laryngology 49

Post-Cricoid Dysphagia with Gastric Symptoms.

By DAN MCKENZIE, M.D.

FOR the last six or eight months the patient, a man, aged 36, has had difficulty in swallowing, together with the sensation of a lump in the throat and tightness, all referred to the post-cricoid level. In swallowing solids, the efforts made resemble those present in post-cricoid obstruction. There is, however, no cough or spluttering with liquids; and food has never been regurgitated. It is noteworthy that salivation was profuse at one period of the history, particularly at night.

On laryngoscopy, some salivary accumulation was noticeable behind the left arytænoid, but the direct tube was passed down the œsophagus without any difficulty, and no change in the mucosa was discovered.

Since first coming under our observation the patient has also been examined at the Royal Free Hospital and elsewhere, and the conclusion arrived at is that he is suffering from hyperchlorhydria and perhaps also from duodenal ulcer.

A Case of Chronic Catarrhal Antral Inflammation.

By L. GRAHAM BROWN, F.R.C.S.

BOY, aged 11, first seen January 4, 1927, with an apparent swelling over right side of nose and superior maxilla, stated to have been increasing for the previous three weeks. Very slightly tender on pressure, and skin somewhat reddened over it. The right nasal fossa was completely occluded by swelling of the mucosa. The left nasal chamber was patent. Transillumination of the antra showed the right side as slightly darker than the left.

The swelling was first thought to be due to some underlying inflammatory focus or new growth. The temperature, however, was normal, and X-rays revealed no evidence of bony growth or apical dental infection.

January 6, 1927.—Radical antral operation: condition typical of chronic catarrhal inflammation of right antrum, as recently described by Dr. Oskar Hirsch in the *Journal of Laryngology*, January, 1927, p. 39.

Condition cleared up as now seen.

Discussion.—Mr. HERBERT TILLEY said he was convinced that chronic catarrh of the antrum was often overlooked, even by specialists. Such cases as the present one were often treated as if they were cases of simple chronic inflammation of the nasal mucosa, and applications of the cautery or other forms of local treatment were carried out. The source of the trouble was a mild, chronic inflammatory condition in one of the accessory cavities. In the fourth edition of his (the speaker's) book, published in 1918, page 201, there was a section devoted to "Chronic Non-suppurative Inflammation" (chronic antral catarrh), in which the symptoms were enumerated and the danger of depending on transillumination was pointed out. In 1925 Harrison wrote that the above was the only text-book which he knew of in this country which emphasized the source of many cases of so-called chronic rhinitis.[1] Sometimes the condition could be cured by a few daily irrigations of the antrum, which might only contain an excess of mucus. When this, or the injection of some mild antiseptic fluid, e.g., argyrol, did not get rid of the trouble, an intranasal drainage should be provided.

He thought it only fair to point out that Dr. Oskar Hirsch did not make the discovery of the pathological condition and its symptoms, since work bearing on it was published in this country ten years ago (*vide supra*).

[1] *Journal of the Newcastle-on-Tyne and Northern Counties Medical Society.*

50 Wharry: *Extensive Lupus of the Upper Air Passages*

Dr. P. WATSON-WILLIAMS said that at an annual meeting of the British Medical Association at Birmingham many years ago, he (the speaker) had drawn special attention to the immense importance of these cases. In some cases none of the recognized symptoms of antral sinus infection were present; the transillumination skiagrams and other diagnostic tests were negative, though by sucking some of the discharge into a syringe from a suspected antrum and culturing it, a definite diagnosis could be made. He had had many other cases in which latent sinus infection was responsible for the patient's troubles, e.g., arthritic and other manifestations were as likely to arise from a sinus infection without signs as from a septic condition in the teeth and mouth. Transillumination was often, in doubtful cases, as misleading as it was helpful; it had no value as a crucial test. When chronic antral inflammation was present, the endorhinoscope would generally reveal streaks of more or less clear mucus, or more likely muco-pus, constantly appearing at the posterior end of the inferior turbinate, the vessels running up to the inferior and middle meatus being usually congested on the infected side. The difference between the sound and the infected side was very striking.

Mr. E. D. D. DAVIS said that in this case there was a swelling and expansion of the right ala nasi suggesting a new growth. It was not a latent sinusitis, as the inflammatory condition of the nose was obvious. At the operation it was found that the polypi had expanded the bony wall of the antrum.

Mr. GRAHAM BROWN (in reply) said that on the first view it looked like a new growth or an inflammatory swelling, possibly due to an apical dental infection. He did not diagnose the condition at first. It was after having made an incision under the lip and entering the antrum that he saw what the condition was. X-rays had shown a definite opacity, and he had therefore proceeded with the radical antral operation.

Extensive Lupus of the Upper Air Passages treated by Radium.

By H. MORTIMER WHARRY, F.R.C.S.

SERIES OF CASES RECENTLY TREATED.

PATIENTS suffering from small lupus patches, which have readily healed after radium applications, are not included in this series, which contains only extensive cases.

The cases have been divided according to the organ principally affected.

(1) *Laryngeal Cases.*

These are cases in which the disease affected the larynx as well as other parts of the upper air passages. These are the most extensive and serious cases as far as the upper air passages are concerned. Owing to the patient's aphonia, the condition of the larynx has been the principal consideration in treatment, apart from actual cure.

Case I.—Types (A) granulations and (C) tumour formation are exemplified. Patient is an unmarried woman, aged 24.

History.—Tuberculosis of apex of right lung which had been quiescent for some years. 1918: Nasal obstruction developed, due to lupus. Wassermann reaction negative. 1919: Voice became husky and disappeared altogether. For four years patient lived the life of a recluse for fear of infecting others, and owing to being unable to speak to anyone.

Examination (June, 1923).—General condition good. Tuberculosis of lungs quiescent. Nose: cartilaginous part bulging as if from large polypi, but in this case caused by extensive crusting within the vestibule. Posterior edges of the vestibule and the anterior ends of the inferior turbinates were covered with large crusts, which also surrounded a perforation of the septum about as large as a threepenny-bit. The

nose was almost completely obstructed by the crusts. On removal of one of the crusts a swollen and bleeding surface was revealed. Post-nasal space: In the right choana, resting against and attached to the septum, was a rounded white tumour about as large as a hazel-nut. Pharynx and fauces: The uvula was rigid and much swollen, with a granular surface. Granulations extended down the posterior left faucial pillar and tonsil bed and both the right pillars and right tonsil bed. The pharynx was heavily scarred from healed lupus. Larynx: Epiglottis scarred, deformed, and apparently healed. Both ventricular bands and vocal cords were thickly covered with granulations, and there was swelling of the interarytænoid region with granular surface. Phonation: The whole larynx was stiff and the cords did not approximate. Voice: None: whisper very feeble and only faintly audible.

Treatment.—Beginning on November 5, 1923, patient received short courses of radium treatment at two-monthly intervals. These were continued for a year.

Result.—After the first application of radium the crusting disappeared from her nose and she was able to breathe through it. Also, within six weeks she found herself able to speak for the first time after a silence of four years. The whole condition has since completely cleared up. Her soft palate was a little stiff, but mobility was restored to this with massage under cocaine anæsthesia by Miss Curling The voice is still somewhat husky, but is loud and the words are clearly audible; she can speak for any ordinary length of time without increased huskiness of voice or tiredness of the throat.

Case II.—Types (A) granulations, (B) vegetations and (C) tumour formation. The patient, a girl aged 18, had for three years suffered from nasal obstruction and complete loss of voice.

Examination.—Nose: Lupus of the vegetating type (B) was protruding from the left nostril. Granulations (Type A) extended through the left nasal fossa as far as the post-nasal space. The posterior half of the right nasal fossa was also covered. Post-nasal space: The posterior ends of both middle and inferior turbinates were affected, also the posterior pharyngeal wall and the superior surface of the soft palate. Pharynx and fauces: The pharynx was much scarred, and the uvula, fauces, and tonsil beds covered with granulations. Larynx: Epiglottis, ventricular bands and vocal cords were all thickly covered. In the interarytænoid region was a fair-sized lupus tumour (Type C). Voice: Completely absent. Whisper only faintly heard. We had the greatest difficulty in persuading this patient or her mother that she would regain her voice. At the Radium Institute two years previously she had been told that the disease was too deep-seated to be attacked by radium.

Treatment.—Courses of radium at two-monthly intervals.

Result.—The whole condition has cleared up except on the right inferior turbinate. There is no nasal obstruction, and no scarring except in the posterior pharyngeal wall and epiglottis. The voice returned in four months and is now quite normal: the patient is able to sing. The palate and fauces are freely mobile and show no scarring.

(2) *Nasal Cases.*

These are cases in which the nasal condition has been predominant, and in which the avoidance of its destruction and disfigurement has been the first consideration in treatment.

Case III.—Shows apple-jelly nodules on nose and lip and Type (A) granulations in the nose and pharynx. Patient, male, aged 18, who had for several years suffered from nasal obstruction, and for one year had noticed spots on the nose and lips, with swelling and crusting of the right nostril.

52 Wharry: *Extensive Lupus of the Upper Air Passages*

Examination.—Nose: In the right nasal fossa the granulations extend back to the naso-pharynx. In the left nasal fossa there is some slight granulation. Naso-pharynx and pharynx: Granulations extend downward behind both posterior pillars of the fauces, which are themselves slightly affected. Larynx unaffected. Neck: both tonsillar glands are swollen. Right lachrymal sac and right eye affected.

Treatment.—The usual line of treatment has been followed.

Result.—The whole condition has made very satisfactory progress. There is now no danger of destruction of the right alar cartilage, and most of the disfigurement at first apparent has disappeared.

Case IV.—Shows apple-jelly nodules on the nose and Type (A) granulations. Patient, a male, aged 21, had for three years suffered from swelling and disfigurement of the nose, with nasal obstruction, and much crust formation in the nostrils. His throat also had been sore and his voice husky for a year.

Examination.—Nose: The whole end of the nose was much swollen and covered with apple-jelly nodules, which also covered the alæ. The vestibule on each side was widely dilated with crusts and granulations which extended far into the nose. There was complete nasal obstruction. Post-nasal space: Some scarring and granulations in the superior aspect of the soft palate. Palate and fauces: The whole of the soft palate and fauces and most of the hard palate were covered with a uniform raised red mass of granulations. Larynx: The epiglottis was thickened with granulations and the anterior end of the left vocal cord slightly affected. The voice was husky but not extremely so.

Treatment.—As in the previous cases.

Result.—The case is still under treatment. The nose is now nearly normal and all risk of breaking down and disfiguration is past. The patient can breathe well through both sides of the nose. The condition of the palate and fauces is rapidly disappearing. The epiglottis is still thickened, but the voice has again become normal.

Dr. P. H. G. GOSSE said treatment of these cases by radium was carried out by two methods. The first was by means of radium screened with 1 mm. of silver, using only the Beta rays, employing a tube like that shown, which would contain 12 mgm., and placed in the nasal or buccal cavity. For treating the inside of the mouth the radium container was put on a wire, and an exposure of two or three hours given.

In the larynx, when the radium could not be applied by the direct method, it was screened with 2 mm. of lead, to cut out all the Beta rays and use only the Gamma, and these radium plates were applied externally so as to bring about a cross-fire irradiation of the whole larynx.

Section of Medicine.

President—Dr. HUGH THURSFIELD.

The Prevention of Scarlatinal Nephritis.

By H. CARTER and A. A. OSMAN, M.R.C.P.[1]

DURING the course of an investigation by one of us (A. A. O.) on the value of alkalis in the treatment of nephritis with œdema, it became evident that apart from their efficacy in this connexion, they also in some measure conferred protection against the deleterious results of certain intercurrent factors which under ordinary circumstances produce an aggravation of a pre-existing kidney lesion or at least an increase of the signs and symptoms with which it is associated. A superimposed infection and over-exertion are the two most important factors which act in this way during the course of a case of chronic nephritis with œdema. Some general anæsthetics and certain drugs, especially metallic compounds such as lead and sanocrysin, sometimes act in like manner, and as is well recognized, should be administered with care in the presence of a damaged kidney. It is not possible here to enter in detail into the rationale of the alkaline therapy of nephritis in general except to point out that in practically all cases of whatever clinical variety, there is a decrease in the plasma bicarbonate and also that the occurrence of any one of the above mentioned conditions in a case of chronic nephritis is invariably accompanied by a still further depletion of this factor. It has been observed that in chronic nephritis with œdema, in which the plasma bicarbonate has been increased to normal by the administration of alkaline salts by mouth, exacerbations due to such intercurrent causes were less severe and more quickly recovered from than in cases not so treated, especially if the disturbing factor were met by a temporary increase in the dose of alkali given. It seemed possible, therefore, that they might possess some prophylactic value in all circumstances under which renal damage was known to exist and under which further impairment of renal function might be expected to ensue. In a few cases in which sanocrysin was being given and in which it had previously produced albuminuria, alkalis given prophylactically by mouth appeared to prevent this complication, and this encouraged us to investigate the problem on a larger scale. Scarlet fever provided at once a ready supply of cases for observation under conditions where the experiment could be carefully controlled and the results observed.

Through the kindness of Dr. H. Woodfield, Superintendent of the Park Fever Hospital, Hither Green, we were able to try the experiment on a comparatively large scale.

Of recent years scarlet fever has been of a very mild type in this country and the incidence of nephritis as a complication very low. It is variously estimated at from 3 to 10 per cent. of all cases. In Scotland this complication appears to be more frequent, whilst on the Continent, at least in some countries, the disease is still of a severe type and nephritis frequently occurs. A doctor from Czecho-Slovakia recently informed us that the present epidemic in that country is very severe and that

[1] Working with the aid of a grant from the Medical Research Council.

44 Carter and Osman: *The Prevention of Scarlatinal Nephritis*

nephritis occurs in approximately 50 per cent. of all cases. The incidence of nephritis and albuminuria in our control (untreated) cases was 3 per cent.

For the purposes of the present research certain wards were set aside in which all cases of scarlet fever were given alkalis as a routine. Patients in the other wards were not given alkalis and were used as control cases. In all other respects, such as diet, clothing, etc., the conditions in the two groups of cases were identical. In the "alkaline" wards in the early stages of the investigation, a standard mixture containing 15 gr. each of sodium bicarbonate and potassium citrate in water with a suitable flavouring agent, was given by mouth to all the patients three times a day, or 90 gr. a day to each patient. This dose seemed to be sufficient to render the early morning specimen of urine alkaline to litmus in most instances in which the patients were on a light diet and confined to bed. Later it became evident that some patients had a higher alkali tolerance and a larger dose had to be given to maintain the morning urine at approximately the same degree of alkalinity. Later it was the custom to give children under seven years of age a daily dose of 200 gr. a day, and to patients over this age up to 400 grains a day. If too small a dose be given the early morning specimen would remain acid to litmus though later samples became strongly alkaline in reaction to this dye. To estimate the degree of alkalinity more accurately, the capillators (B.D.H.) were used. During the earlier months of the investigation alkalis were given for the first twenty-one days in hospital only, but as this resulted in two cases of nephritis developing twenty-four hours after the medicine had been withdrawn, the procedure was afterwards continued throughout the course of the illness. No ill-effects were observed in any of the cases so treated; on the contrary, the impression obtained was that these patients were, generally speaking, more comfortable, and suffered less from complications such as "rheumatism" than the controls. Statistics dealing with this aspect of the subject are at present being collected.

The following tables show the incidence of nephritis and albuminuria in the patients treated, and in the controls, over a period of fourteen months:—

TABLE I.—CASES TREATED WITH ALKALIS.

Age incidence	No. of cases	Nephritis	Albuminuria
Under 3 years	9	0	0
3— 4 years	44	0	0
4— 5 ,,	112	3 (1 fatal)	0
5—10 ,,	344	1	0
10—15 ,,	60	0	0
15—20 ,,	32	0	0
20—25 ,,	12	0	0
Over 25	7	0	0
	620	4	0

Incidence of Nephritis = 0·6 per cent. Albuminuria = 0.
Total incidence = 0·6 per cent.

It will be observed that in this group there were four cases of nephritis. Three of these were mild cases in which blood and albumin appeared in the urine forty-eight hours after the treatment had been withdrawn, as explained above. In the interval between the cessation of treatment and the appearance of the nephritis, the urine in each case had become acid (pH 5·0, 4·4, and 4·8). In the fourth and fatal case, that of a female child, aged five years, nephritis developed on the fourteenth day of the disease whilst the patient was receiving alkalis (pH 7·5). Apart from passing 2 oz. of urine two days later, she had complete suppression until death, which took place nine days from the onset. Sections of the kidneys from this case were examined by Dr. J. E. McCartney, and also by Professor

Adrian Stokes, of Guy's Hospital, and both reports agreed that a previous nephritic lesion existed. Dr. McCartney's report stated that the section showed evidence of subacute interstitial nephritis with antecedent glomerular change. Incidentally it was ascertained that none of the cases in the "alkali" group developed albuminuria or nephritis after being transferred to the convalescent hospitals.

TABLE II.—CONTROL CASES FROM THE WARDS IN WHICH ALKALIS WERE NOT GIVEN.

Age incidence	No. of cases	Nephritis	Albuminuria
Under 3 years	104	1	1
3— 4 years	51	0	0
4— 5 ,,	27	1	1
5—10 ,,	61	1	1
10—15 ,,	40	0	1
15—20 ,,	8	0	0
20—25 ,,	14	1	1
Over 25	11	0	0
	316	4	5

Incidence of nephritis = 1·3 per cent. Albuminuria = 1·6 per cent.
Total incidence = 2·9 per cent.

TABLE III.—CONTROL CASES FROM A HOSPITAL SERVING THE SAME AREA AS THE PARK HOSPITAL, AND COVERING THE SAME PERIOD.

Age incidence	No. of cases	Nephritis	Albuminuria
Under 3 years	26	1	0
3— 4 years	28	0	1
4— 5 ,,	40	3 (1 fatal)	3
5—10 ,,	144	1	2
10—15 ,,	50	1	2
15—20 ,,	26	1	3
20—25 ,,	11	0	1
Over 25	11	0	0
	336	7	12

Incidence of nephritis = 2 per cent. Incidence of albuminuria = 3·5 per cent.
Total incidence = 5·5 per cent.

Though these figures cannot in any way be considered final it is submitted that alkalis given by mouth in the amounts used in this investigation do appear to have some prophylactic value in the prevention of scarlatinal nephritis. Individual variations as to "alkali tolerance" have so far made it impossible to state with certainty a minimum standard dose which will at once be safe and completely preventive in all cases. Further observations on the urinary pH and "alkali tolerance," under various conditions and in the presence of associated factors such as sepsis and pyrexia, may lead to even more complete protection from this complication.

We take this opportunity of expressing our indebtedness to Dr. H. Woodfield for his kind help and for giving us the facilities for carrying out this investigation. Our thanks are also due to the Medical Superintendent of the "Control" Hospital for the figures supplied to us, and also to the Sisters and Nursing Staff at the Park Hospital for their invaluable assistance.

Discussion.—Dr. J. D. ROLLESTON emphasized the very low incidence of nephritis in scarlet fever in recent years. At his own hospital (Western Hospital) he had not been able to show his students a single case in the course of six months, and he had had barely six cases of nephritis among over a thousand scarlet fever patients in the course of a year.

46 Carter and Osman: *The Prevention of Scarlatinal Nephritis*

The experience of other medical superintendents of the Metropolitan Asylums Board Hospitals with whom he had discussed the matter had been similar. Foreign observers such as Brelet [1] at Nantes, Lefebvre [2] in Paris, and Steiner [3] at Budapest, had also drawn attention to the rarity of nephritis in scarlet fever in recent years. Under the circumstances it appeared doubtful whether the method in question was really the cause of the very slight difference in the incidence of nephritis among the cases treated with alkalis as compared with the controls. The authors had not stated what preparation they had employed, but it probably would not have the injurious result inherent in the only other prophylactic drug treatment of scarlatinal nephritis known to him, namely, administration of urotropin, which, as Steinitz had shown, [4] was liable to cause cystitis. In conclusion he (Dr. Rolleston) was inclined to regard the method as an example of the *nimia diligentia medici* to which Sydenham alluded in his chapter on scarlet fever.

Dr. F. PARKES WEBER said he thought that what occurred in ordinary persons when they took excessive and unaccustomed muscular exercise might be compared with what occurred when katabolic metabolism was suddenly increased owing to the commencement of a febrile infection. In both cases the urine became very acid and concentrated, and yielded an abundant sediment of urates on standing, and in both cases there was an unpleasant subjective sensation of pain or painful stiffness in the muscles and associated fibrous and connective tissue. He (Dr. Weber) believed that as sodium bicarbonate and similar drugs helped to relieve the unpleasant results of excessive muscular exercise they were probably also beneficial at the commencement of infectious fevers. In regard to urotropin he thought that if it were tried in the hope that it would prevent the development of scarlatinal nephritis, it should be administered in association with large doses of sodium bicarbonate solution, in order to avoid irritation of the urinary bladder.

Dr. J. KINGSTON BARTON said that the modern pathologists described three forms of nephritis that might occur in scarlatina: (1) Pure interstitial nephritis in the very early days of the fever; (2) glomerular nephritis in the middle stage of the fever, and (3) a true tubular nephritis in the desquamative stage towards the end. If the pathologists could tell us what element in the disease, the bacterium or the toxin, it was which caused the onset of the different types of nephritis, the rationale of the administration of alkalis or any other type of drug as a preventive would be better understood. In typhoid fever it was well known that the bacteria actually passed through the renal system and were eventually killed by the administration of urotropin, without any damage to the kidneys. But, as Dr. Rolleston had pointed out, in scarlatina this drug was positively harmful. Between the years 1880 and 1890 scarlatina of a severe type was very prevalent in London, and it was the custom in those days to try to prevent nephritis by giving liquor ammonii acetatis in order to keep the skin moist and thus favour less elimination by the kidneys. In addition citrate of potash or sodium bicarbonate with fresh lemon juice was given to keep the urine less acid and more bland; and without our knowing it acidosis (a term not used in those days) was combated. The author's tables showed that there were cases of albuminuria without signs of any type of nephritis, and suggested that unless there had actually been some mild form of nephritis, the albuminuria must be ascribed to cardiac weakness.

1 *Journ. de Méd. de Paris*, 1926, xlvi, 1051.
2 Thèse de Paris, 1926, No. 563.
3 *Jahrb. f. Kinderheilk.*, 1927, cxv, 348.
4 *Monatsschr. f. Kinderheilk.*, 1923, xxv, 617.

Section of Neurology.

President—Sir James Purves-Stewart, K.C.M.G., C.B., M.D.

Optic Nystagmus.

By E. Arnold Carmichael, M.R.C.P.

There are six or seven clinical types of nystagmus: congenital nystagmus, spontaneous nystagmus, miners' nystagmus, vestibular nystagmus, nystagmus of central origin, and nystagmus of cerebral origin. The pathological conditions in which nystagmus may be elicited are manifold, but they may be divided into seven groups according to the supposed site of the causal lesion: (1) high spinal lesions—above the fourth cervical segment; (2) medullary lesions; (3) cerebellar lesions; (4) vestibular lesions; (5) mid-brain lesions; (6) possible lesions of the retina, and (7) certain congenital anomalies, such as albinism. In the symptomatology of these lesions a diagnostic feature is nystagmus, which is readily produced on voluntary deviation of the eyes to the right or left.

I am about to deal with a type of nystagmus which can only be evoked in normal individuals by special methods and which in certain pathological states cannot be elicited. In 1907 Bárány drew attention to a form of nystagmus which he described as "Eisenbahn" nystagmus. Since then there have been many papers upon this form of nystagmus, chiefly written by ophthalmologists, neurologists, and otologists; but so far as I have been able to ascertain, nothing has been recorded of the physiological or experimental work on the subject.

On a railway journey it is a common practice to note the presence of a nystagmus in the eyes of the passenger seated opposite, who is viewing the passing landscape through the window. Various names have been given to this nystagmus—"railway nystagmus," "optomotor nystagmus," "optic rotation nystagmus," and "optic nystagmus." This diversity of terminology has caused considerable confusion in the literature, especially in German literature. In this country Fox and Holmes, who have done much clinical work upon the subject, have named it "optic nystagmus," and in this paper I shall adhere to their terminology.

It was not until 1913 that Bárány applied this test for diagnostic purposes in clinical medicine. He constructed a drum upon which he drew either vertical stripes or various figures of pictorial interest. This drum was held with a vertical axis in front of the patient's eyes and rotated in a clockwise, or anti-clockwise direction. This rotation immediately evoked a nystagmus which consisted of two components—a slow phase and a rapid phase. On rotating the drum in a clockwise fashion so that the figures passed from the right to the left of the patient, optic nystagmus was produced with the following characters: The slow phase was in the direction of the rotation of the drum to the left, while the rapid phase was to the right. Thus with the drum rotating clockwise, a nystagmus to the right was obtained (a nystagmus being designated according to the direction of the rapid component), vice versa, with the drum rotating anti-clockwise, an optic nystagmus to the left resulted.

Obviously absence, increase, or inversion of this nystagmus may prove of value

in clinical diagnosis. Unfortunately, the physiological work upon this subject has not as yet localized the centres and tracts connected with its production. Therefore one is forced to attempt to find an "arc" for its production from a study of the recorded cases in which it has been absent to one side or the other and in which definite localized lesions have been present. Recently Fox and Holmes have published a paper of great clinical value on this subject. Cords, Wernoe, Strauss, Stenvers and Ohm—the last a most prolific writer—have recorded many observations upon patients in whom there was a failure to produce the nystagmus.

As it is impossible to produce the nystagmus in people totally devoid of vision, it is obvious that some sense of vision must remain, though only in a minor degree. One is therefore led to assume that the optic nerves, tracts and radiations, as far as they are concerned with the transmission of stimuli from the eye to the occipital cortex, must in some degree remain functioning. Let us examine lesions of the optic sensory path to the occipital cortex. There are reports on record of patients suffering from severe and large central scotoma, with very little peripheral vision left, in whom it was still possible to produce an optic nystagmus. Further, there are other cases on record which show that patients, with one eye blind and only a small part of the peripheral field of vision of the other eye left, have an optic nystagmus remaining to both sides. This therefore suggests that lesions of the optic nerve or chiasma in no way interfere with the production of the optic nystagmus, provided some peripheral vision remains.

Cords reports upon a case in which a homonymous hemianopia was present, due to a lesion of the optic tract, and in which the optic nystagmus was absent. He found the optic nystagmus, as designated by its quick component, to be absent to the side opposite to the lesion—thus a right optic tract lesion caused a loss of the optic nystagmus to the left. But the series of cases reported by Fox and Holmes have not borne out this observation, and we may therefore assume that a lesion of the optic tract does not necessarily cause the loss of the optic nystagmus to the opposite side. From the region of the geniculate bodies—the end stage of the first neurone—originates the second neurone, which passes to the occipital cortex. This second stage in the pathway of visual stimuli involves tracts passing from the region of the geniculate bodies through the posterior limb of the internal capsule by way of the optic radiations to the occipital cortex. It is therefore important to discover whether lesions causing a break in this neurone will result in the absence of optic nystagmus to one side or the other.

Difficulty is immediately encountered, as lesions affecting this tract never occur without damaging other sensory, motor, or association tracts. In cases in which a homonymous hemianopia has occurred, due to a supposed lesion on this path, optic nystagmus has still been present, even to the blind side. Bárány, however, in 1913, described two cases of hemianopia in which optic nystagmus to the blind side was absent and he suggested that the lesion was in the region of the optic radiation. Fox and Holmes, Ohm, Stenvers, and Cords have recorded cases proved to have been suffering from a lesion of the radiation resulting in varying degrees of hemianopia. As several of these showed loss of the optic nystagmus to the hemianopic side one is forced to assume that a lesion affecting the optic radiations and causing hemianopia may cause the loss of optic nystagmus to the opposite side.

No case with a unilateral lesion in the occipital lobe is recorded in the literature. Cords reports upon a patient in whom he believed the occipital cortex on one side to be totally damaged and on the other side only partially damaged. In this case optic nystagmus was absent on the side with complete hemianopia—that is, opposite to the side of the lesion. Fox and Holmes, in their series of cases, found that a lesion in the region deep to the supramarginal gyrus, with or without hemianopia, but

frequently associated with hemianopic inattention, caused a loss of the optic nystagmus to the blind or inattentive side. Thus a lesion in the left supra-marginal gyrus may cause absence of the optic nystagmus to the right.

Stenvers also published a series of cases in which lesions situated in the region of the posterior end of the Sylvian fissure, either associated or unassociated with hemianopia, caused a loss of the optic nystagmus to the opposite side. Therefore, lesions situated in the region of the higher visual centres are frequently associated with loss of the optic nystagmus to the opposite side. Cords describes a patient, who was right handed, with right sided hemianopia and alexia, in whom the optic nystagmus was lost to both sides. Post mortem, a tumour was found in the region of the splenium infiltrating the left half of the brain more than the right. This is the only case I have found in the literature with optic nystagmus absent to both sides, in which a cerebral lesion was undoubtedly the cause.

Lesions of the frontal lobe, especially if situated in the region of the motor area of the eye, cause a loss of optic nystagmus to the opposite side.

Having surveyed the various cerebral lesions located on the visual pathway and in the motor cortex which affect the production of optic nystagmus, it is of interest to know what may occur in lesions situated at a lower level. Reports are on record of patients with mid-brain lesions who had a loss of conjugate deviation of the eyes to one side. On the attempt being made to elicit the optic nystagmus it was found impossible to produce it towards the side to which the power of voluntary deviation was lost; but, to the opposite side, in the place of a nystagmus, a painful and prolonged deviation of the eyes took place.

Ohm has attempted to elicit optic nystagmus in all types of lesions affecting the visual apparatus. He has found that, in cases of miners' nystagmus, three things may happen:—(1) No change. (2) Slowing down of the nystagmus. (3) Miners' nystagmus and optic nystagmus amplifying each other. In cases of retinitis pigmentosa and amblyopic nystagmus, he found that, instead of the optic nystagmus being in the opposite direction to the rotation of the drum, the quick component coincided with the direction of rotation. In other words there was a definite inversion of the normal optic nystagmus. The effect of lesions of the vestibular apparatus and cerebellum is only briefly referred to in the literature. Statements are made that variation in the amplitude, rate and duration of the optic nystagmus may occur, but no definite data are published. From a study of the cases reported in the literature in which the optic nystagmus has been absent it is permissible to map out the probable pathways involved in the production of optic nystagmus.

It is not necessary for there to be an intact optic nerve, chiasma or tract; further, an intact optic radiation is not essential. Thus, by deduction, it may fairly be assumed that with only one visual sensory path to the occipital cortex it is possible to obtain an optic nystagmus to both sides.

It is certain that lesions "higher" than one occipital cortex cause a loss of the optic nystagmus to the opposite side. It is, therefore, probable that from the occipital cortex an association bundle passes to the higher visual cortex on the same side; further, that the occipital cortex on one side is connected through the splenium with the higher visual centres of the opposite cerebral hemisphere. A lesion affecting one or other of these association tracts may cause a loss of the optic nystagmus to the opposite side. Further, a lesion of the splenium which damages the association tract connecting the two occipital lobes causes a loss of the optic nystagmus to both sides.

There is also in all probability an association bundle connecting up the higher visual centres of one side with the eye centres in the pre-Rolandic gyri of the

same side—a breaking of this tract causes a loss of the optic nystagmus to the opposite side.

Lastly, damage to the pre-Rolandic centre interferes with the production of the optic nystagmus to the opposite side.

In short, the possible pathway of the reflex is as follows: along the optic tract and optic radiation to the occipital cortex; from there to the higher visual cortex whence a bundle passes to the pre-Rolandic region, from whence stimuli are sent to the oculo-motor nuclei.

Stenvers put forward the hypothesis that reflex centres for optic nystagmus lie in the occipital lobe and in the second frontal convolution, these centres being connected by an association bundle passing through the substance of the white matter. Cords suggests that the path for the production of optic nystagmus is as follows: The sensory portion is by way of the optic nerves, tracts and radiations, back to the occipital cortex; from thence impulses pass to the visual motor cortex immediately in front of the occipital lobe and through the splenium to the opposite side, from which an impulse is carried to the motor cells in the region of the fourth nucleus on the opposite side. Cords supposes this impulse to travel by way of the optic radiations down through the internal capsule to the oculo-motor nuclei which are also controlled by a path from the opposite frontal lobe. He therefore states that lesions on the sensory path do not cause loss of the optic nystagmus, but lesions affecting either the visual motor cortex or the motor path from the cortex to the oculo-motor nuclei do cause this loss.

The phenomenon of optic nystagmus may be easily produced in normal individuals; there is a record of its production in an infant eight hours old. This suggests that neither myelination nor power of fixation is necessary for its production. In order to elicit information regarding its nature several normal individuals have been examined, and to estimate the sensitivity to the production of optic nystagmus a series of experiments were carried out, as follows: A hoop with a circumference of approximately 64 in. was suspended by a long thread to a stand so that it would rotate on a vertical axis at an even rate for a considerable space of time. To the outer side there was attached a strip of paper having alternate black and white vertical stripes, 2 in. wide. By rotating this instrument it was easy to produce optic nystagmus. For the purpose of these experiments the subject was placed with his back to the window and the striped paper on the hoop exactly 2 ft. from his eyes: thus the width of exposed paper remained constant.

Using varying speeds of rotation one was able to produce an optic nystagmus which varied in its characteristics. Clockwise rotation of the hoop always produced a nystagmus to the right and anti-clockwise a nystagmus to the left. With the stripes passing before the eyes at a rate of about 120, or sixty black stripes, each minute a nystagmus with a rate of seventy-five per minute was produced. As the stripes were made to rotate more rapidly the nystagmus became more frequent. When the rate of rotation became fast an irregular nystagmus was produced; eventually with very fast rotation no nystagmus was produced. When questioned about the subjective sensation resulting, each subject said that he could not voluntarily control the nystagmus except by staring and that a stage arrived at which the stripes became a blur. The point of interest which came out unexpectedly was that instead of there being a sudden cessation of the nystagmus upon the increase of rate of the stripes, there was a gradual irregular diminution in the rate of the nystagmus.

By plotting out the number of nystagmoid jerks and the various speeds of rotation one obtained a curve, which showed a fairly steady rise, a long broad plateau, and a rapid descent. In a series of experiments with healthy, young adults, the curves were almost identical in the same subject for both directions of

rotation. A comparison of the curves for various individuals showed a marked resemblance. While this work was being done an interesting observation was made. It was noticed that when the speed became very rapid the eyes remained deviated towards the edge of the hoop where the stripe disappeared.

Again, the same hoop being used, screens were intervened between the subject's eyes and the stripes, so that varying lengths of striped paper could be exposed to their view. Upon 2 in. or less of the striped paper being exposed, no nystagmus was produced. But when the opening was widened to 2¼ in. a fine nystagmus was produced. The width of striped paper that had to be exposed to obtain the nystagmus varied in different individuals; nystagmus was never obtained with a field of view of less than 2 in., and in one case not until a gap of 2½ in. had been made. Therefore, in order to obtain the nystagmus, two stripes must be in sight at the same time. This rather appears to uphold the view of Bárány that the quick phase was the result of a quick glance back to catch the oncoming stripe.

Further, it was observed that the amplitude of the nystagmus varied with the width of the exposed striped paper. A very fine nystagmus was obtained with narrow widths of exposure; whilst the greatest amplitude was obtained with a width of 3½ to 4½ inches of exposed striped paper. This was found to vary in several apparently healthy individuals, but in the one individual the width remained constant for both clockwise and anti-clockwise rotation.

As more than one individual upon whom these experiments were performed complained of a feeling of giddiness it was thought that there must be some relation between the vestibular apparatus and the centres connected with the optic nystagmus. An attempt was made to reproduce vestibular lesions by rotation or by irrigation of the ear with hot or cold fluid, and to observe the effect upon optic nystagmus. It is well known that should the right ear be irrigated with cold water a nystagmus will be produced, with a slow component to the right and a rapid component to the left. And it has been proved that should this be done under an anæsthetic a steady deviation of the eyes to the right will result. This last fact points to the slow component of the nystagmus as being of undoubted vestibular origin. Pike, working from a physiological standpoint, found that decerebration of animals had no effect upon the magnitude of the slow movement in vestibular nystagmus, as produced by rotation or irrigation. Secondly, he found that if one cerebral hemisphere was removed this abolished the quick movement when the slow movement of the eye was directed to that side, but did not affect the quick movement when the slow movement of the eye was directed to the remaining cerebral hemisphere. Thirdly, he found that the quick movement of the eyes was abolished when only the temporal and basal portions of the cerebral hemisphere of the one side were removed just as when the whole hemisphere was removed. He therefore suggested that the quick phase in vestibular nystagmus was of cerebral origin. Ivy, however, found that decerebration of animals had no effect upon the quick component of the vestibular nystagmus, provided that the temperature of the animal was kept constant. Magnus, working more recently, elicited nystagmus by caloric and rotation tests after the brain-stem had been divided caudally to the trochlear nerves, and from his work he came to the conclusion that the quick phase in vestibular nystagmus was in no way dependent upon a cerebral pathway. It is therefore apparent that vestibular nystagmus differs entirely from optic nystagmus; for the quick and slow components in optic nystagmus require the functioning of certain portions of the cerebral hemispheres. Now if one irrigates the left ear of a healthy individual with ice-cold water for a minute and a half, a vestibular nystagmus will be produced which can be elicited on deviation of the eyes laterally. On deviation to the right there will be a fine rapid nystagmus with the slow phase to the left; and on deviation to the left

there will be a nystagmus of greater amplitude and more difficult to obtain, but with similar components. After such a vestibular nystagmus had been produced in a healthy individual the subject was immediately tested for optic nystagmus. With the drum moving in an anti-clockwise direction, which should give an optic nystagmus to the left, no optic nystagmus was produced; on rotation of the drum in a clockwise direction an optic nystagmus was obtained with greater amplitude than when the subject was tested previously to the irrigation of the ear. Upon one individual being constantly tested by simple voluntary deviation of the eyes laterally a time came when the vestibular nystagmus ceased to be present; but upon his being tested at this stage for optic nystagmus it was found that the optic nystagmus to the side of the ear irrigated with cold water was still in abeyance. It would, therefore, appear that not only does a vestibular disturbance over-rule an optic nystagmus, but that it continues to do so after all clinical tests to establish the presence of a vestibular nystagmus have become negative. We have thus, in optic nystagmus, a very delicate method for discovering slight dysfunction of the vestibular apparatus and, further, a method which is practically devoid of the unpleasant sensation and sequelæ associated with the irrigation and rotation tests. Upon other such experiments being carried out it was found that the width of stripes exposed affected the delicacy of the test; and as a result of a series of tests it became evident that a width of 4 inches was most suitable for use as a standard width.

It was next attempted to find out the effect of rotation tests upon optic nystagmus. After rotation of an individual ten times in a clockwise direction, stripes passing in a clockwise direction produced a rapid nystagmus of fair amplitude with the rapid phase to the right, while with stripes passing in an anti-clockwise direction a nystagmus was produced of greater amplitude with the rapid phase still to the right. Thus an inversion of the normal optic nystagmus to anti-clockwise rotating stripes had taken place. It therefore appears reasonable to assume that stimulation by rotation of the vestibular nerve on both sides at the one time causes a complete disturbance of the optic nystagmus. It was found impossible to demonstrate any change in the optic nystagmus after the cessation of the vestibular nystagmus as demonstrated by voluntary deviation of the eyes laterally.

As yet, an opportunity for examining patients with diseases of the vestibular apparatus has not presented itself, but in the light of these experiments it would appear that an attempt to produce an optic nystagmus in such patients may yield interesting results.

Conclusions.

A résumé of the work upon optic nystagmus in healthy individuals and in disease warrants the following conclusions:—

(1) That optic nystagmus is lost to the opposite side when a lesion is situated:—(*a*) in the higher visual cortex of one side; (*b*) in the frontal lobe of one side; (*c*) in the association tracts connecting the higher visual centre and the frontal lobe of the same side.

(2) That optic nystagmus may or may not be absent in lesions of the optic radiations.

(3) That optic nystagmus is unaffected by lesions of the optic tracts, optic chiasma and optic nerves, provided some sight remains.

(4) That optic nystagmus is dependent upon a pathway situated in the cerebral hemispheres.

(5) That optic nystagmus is affected by disorders of the vestibular apparatus.

(6) That slight vestibular dysfunction may be recognized by changes in the optic nystagmus.

Section of Neurology 59

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Demonstration of Clinical Measuring Instruments.

By F. C. EVE, M.D., F.R.C.P.

THE most important piece of teaching I carried away from Cambridge was from Sir Napier Shaw, who impressed on me that the quantitative study of a subject is always much more fruitful than any mere qualitative investigation. This principle has not yet been applied to clinical work nearly so far as is desirable.

I can only speak as a general physician who has attempted slightly to remedy this deficiency and to devise a few quantitative clinical instruments.[1]

Strangely enough, I found the most virgin soil in the field of neurology.

(1) TESTING OF THE SENSATION FOR PIN-PRICKS.

This is still done by the crude method of pushing a pin against a patient. There is no guarantee that the force used is the same on both sides. Neither can the same stimulus be reproduced after a week or a month.

This simple instrument—merely a needle mounted in a tube on a long spring—produces practically the same stimulus each time (fig. 1). Of course it is not enough to prick once on each area: a group of three or four pricks at each place is necessary, so as to test an average sample of skin.

FIG. 1.—Sensation tester.

Similarly, in testing for touch, it is easy with cotton wool to produce unequal stimuli. The other end of this instrument consists of a camel's hair brush with parallel fibres. When pushed vertically against the skin, the fibres will always bend and yield to the same force. Hence there is the same summation of little pushes each time. Presumably this is a test of the sensation of touch; certainly it is not felt as a prick. If this is a proper test of touch, it seems a more accurate and less complicated test than the usual sliding contact of cotton wool. Of course the brush contact can be sliding, if preferred.

For desk work I rather prefer the extra length of the combined instrument, but for the pocket it unscrews into two parts.

(2) MEASUREMENT OF WASTING IN LIMBS.

My next instrument is designed for measuring wasting in limbs. Everyone knows the impracticability of pulling the tape measure with the same tension in the two limbs. Hence accurate measurement is impossible with the ordinary tape. An error of a quarter inch or more is common. Still more hopeless is it to reproduce the

[1] For the loan of the blocks illustrating the instruments described, the author is indebted to Messrs. Down, Bros., Ltd.

60 Eve: *Demonstration of Clinical Measuring Instruments*

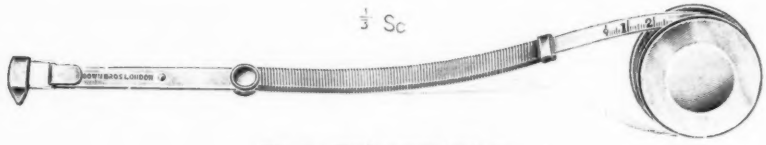

FIG. 2.—Flesh measuring tape.

same tension in the tape after a week or a month when one wishes to discover whether the wasting is diminished or not. The spring tape-measure I show (fig. 2) will effect these requirements. The weight of the spool is equal to the pull of the spring when extended up to the stop, so that the same tension in both ends of the tape is always exerted—whether you are comparing the two limbs or the same limb in the present, past and future. The measurements obtained will be consistent within 1 or 2 mm. A linen tape can be made if preferred, for steel tapes are always fragile.

The instrument should also be very useful to masseurs.

(3) THE WHEEL GONIOMETER.

This instrument is employed partly for the quantitative measurement of Kernig's sign and partly for measuring the stiffness of joints (fig. 3).

It consists of a graduated wheel and a long arm to set along the leg, and will indicate the angle between the leg and the spine. But the angle is useless unless the force used in flexing the leg is constant. This is secured by lifting the lower calf and noting the angle at the hip at which the knee automatically flexes. When the patient has been made to relax his knee, this angle at the hip-joint is consistent and is recorded daily as Kernig's angle. As the wheel is weighted, the

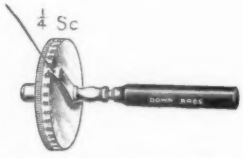

FIG. 3.—Wheel goniometer.

zero is always uppermost. Hence there is no need to burrow into the bed or to find the hip-joint; the axis of the leg anywhere will suffice. To measure the angle of stiff joints a second arm is inserted into the wheel of the instrument. Then it is obvious how it measures the range of movement of a stiff joint. Hitherto it has been very difficult to measure pronation and supination angles. With the use of this instrument this becomes perfectly easy; the patient holds it in his hand horizontally and pronates and supinates. The two angles are read off direct.

Kernig's Angle—Illustrative Case.—Child aged 12, with fourteen days' otorrhœa; admitted delirious with fever, stiff neck and left Babinski. Cured by 42 gr. of hexamine daily.

	Kernig's angle	
	Right Degrees	Left Degrees
February 1, 1924	135 ...	135
,, 4 ,, ...	120 ...	120
,, 9 ,, ...	110 ...	115
,, 12 and 16 ,, ...	110 ...	110
,, 19 ,, ...	100 ...	100
,, 26 ,, ...	90 ...	90 (cured)

(4) CEREBRO-SPINAL MANOMETRY.

This I have practised regularly since 1903 when I brought my old instrument into use. This manometer often affords useful information otherwise unattainable. Sometimes a high pressure is the only abnormality detectable. The cerebro-spinal pressure should always be measured when lumbar puncture is performed medically. There is no extra trouble required with the employment of this instrument I have recently brought out. It is simple and portable, and accurate enough for clinical purposes. If the fluid rises in the tube over a foot, something wrong inside the skull is indicated. To that rule I have found only one exception in twenty-four years, and that was in the case of a patient wildly delirious with apical pneumonia. A rise of 10 in. usually denotes some pathological conditions. The same rubber tube enables one to regulate the flow to any speed required. You merely hold the outflow end higher or lower. You will then see how you are reducing the pressure and can stop the flow at any desired pressure. Even with cerebral tumours I have experienced none of the alleged direful results, the occurrence of which, however, I do not go so far as to deny. But recumbency, and a slow outflow regulating the pressure, must minimize the risks. The use of this needle has other advantages which I cannot dwell upon now.

To perform a lumbar puncture without measuring the pressure is just uninformed laziness. But to omit to measure and then talk about the pressure causing this and that symptom is scientifically most reprehensible. The rate of outflow is no safe guide to pressure; I have often found a high pressure and slow outflow.

(5) TECHNIQUE OF CISTERNA PUNCTURE.

The present technique leaves much room for improvement. There is no method of predicting the depth at which fluid—or the medulla—is to be anticipated. In the case of children one had no guidance at all. We were also told to aim at the glabella which could not be seen or at a pencil in the ear which is in the wrong plane. The mid-line was easy to miss and hence the thecal canal. Naturally my first endeavour was to find some way of predicting the depth at which fluid would be struck. The circumference of the neck divided by nine I found gave a sufficiently accurate measurement both in children, infants and adults, whether fat or thin. Lately I have been trying the ear-to-ear measurement (via sub-occiput) divided by five, but am not yet certain whether this is more accurate and reliable. In the only four cases tried it was very accurate.[1]

The Direction Guide. (Fig. 5).—This is a C-shaped rigid iron frame carrying a nose-piece at one end and two parallel guides at the other. The guides are fixed so that they always point at the nose-piece. The nose-piece and ear-piece are adjustable. The needle is made to enter between the two guides and is kept parallel with them till its direction is fixed by the flesh. Then the guide is removed with the assurance that the needle is in the mid-line and is also aiming at the root of the nose. It is due to our trustful patients that no available safeguards shall be omitted from this hazardous procedure.

With the employment of this technique, one can almost guarantee the certainty of: (1) predicting the safe depth; (2) of not overshooting that depth accidentally; (3) of aiming your needle right; (4) of knowing directly your needle has reached fluid.

Hence the risks seem reduced to almost negligible proportions, and the pain experienced is not great.

[1] In a later case—that of an infant with a very broad head—this ear-to-ear measurement was too great, but the neck divided by nine was accurate.

62 Eve: *Demonstration of Clinical Measuring Instruments*

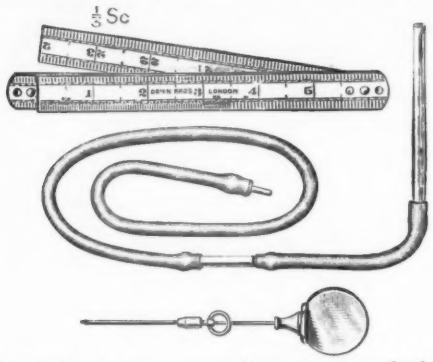

FIG. 4.—Lumbar puncture needle, monometer and rule.

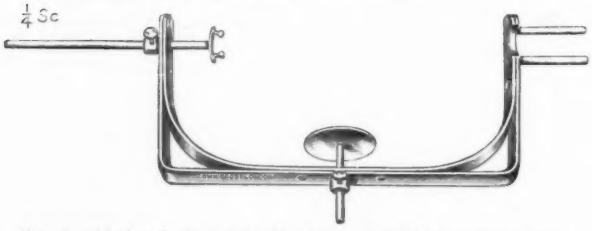

FIG. 5.—Aiming device, with adjustable nasal bridge and aural rest.

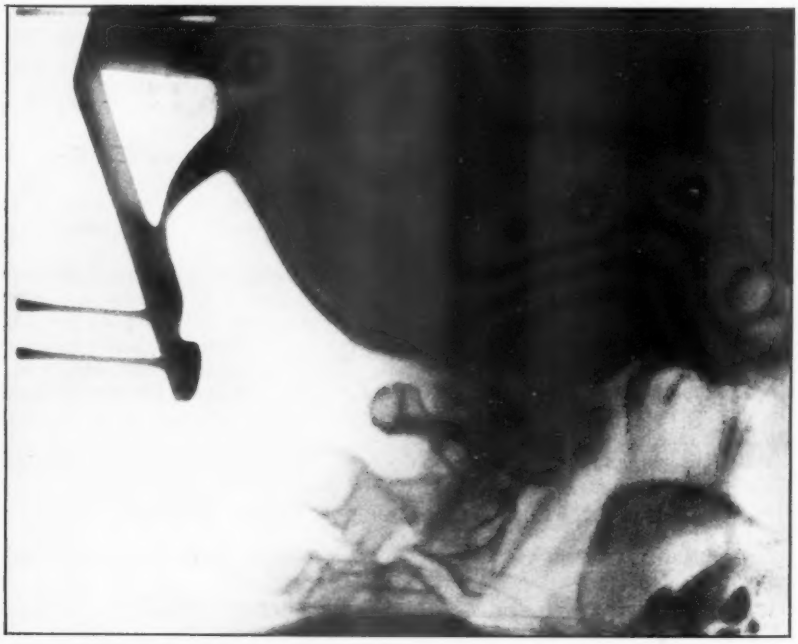

FIG. 6.—"Skiagram (lateral view) of region of cisterna magna with guide in position, demonstrating that a needle passed through the arch parallel to the guides must enter the cisterna. The skin, on which the arch rests, is not shown."

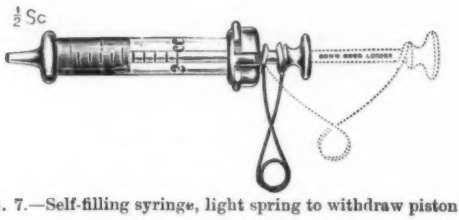

FIG. 7.—Self-filling syringe, light spring to withdraw piston.

DEPTHS FOR CISTERNA PUNCTURE—PREDICTED AND FOUND.

Actual Punctures

Fluid found at	Neck 9	Ear to ear 5
2·5 cm.	2·3 cm.	—
2·8 ,,	2·4 ,,	—
3·0 ,,	2·5 ,,	—
3·5 ,,	3·3 ,,	3·4 cm.
4·5 ,,	3·8 ,,	4·6 ,,

Post-mortems.

3·2 cm.	2·8 cm.	—
2·4 ,,	2·2 ,,	—
4·0 ,,	4·2 ,,	—
5·0 ,,	4·4 ,,	—
3·8 ,,	3·6 ,,	—
3·0 ,,	2·8 ,,	—
3·3 ,,	3·2 ,,	—
4·4 ,,	4·1 ,,	4·7 cm.

My results need checking by those favoured with more opportunities. The neck-girth divided by nine is a conservative estimate, so one may often exceed it by ½ cm. cautiously.

The Safety Button.—This device grips the fragile needle gently but firmly by means of three lead shot pressed convergently by a spring. It cannot be moved unless one pulls on a ring. Hence you set the button on the needle at the depth (predicted by the neck-girth) which you do not intend to exceed. As the occipito-atlantoid ligament in adults is often tough, one feels very much safer with the use of this button, which prevents the needle from over-shooting its mark. Water sometimes lubricates the shot too much, so cautious flaming is better for sterilizing it. Or, if preferred, a chain of metal beads on the needle (one of which is detachable) may be used.

The Self-filling Syringe. (Fig. 7).—It sometimes happens, especially in the sitting-up posture, which is indispensable in lipiodol injection for spinal tumour—that the fluid does not come out, although the needle has entered the cisterna. Once I experienced a delay of over a minute before fluid appeared. The temptation then is to push the needle too deep. I eliminated this risk by making a syringe which fills automatically. When you think you have gone almost deep enough, you remove the stilette and substitute this self-filling syringe with its plunger pressed down. Advance the needle cautiously; directly the point enters the cisterna the syringe will indicate this fact by filling itself with fluid. I have not tried this device often enough to be sure of its value; I believe, however, it will prove useful.

Discussion.—SIR JAMES PURVES-STEWART (President) said that he liked the sensation tester, but thought that its spring should be stronger. He also admired the ingenuity of the instruments and thought the round flat handle of the lumbar puncture needle the best he had used. He was not convinced that the depth at which fluid would be struck could be predicted

64 Eve: *Demonstration of Clinical Measuring Instruments*

by Dr. Eve's methods. He thought the pencil in the ear was a sufficient guide without the aiming device, and said the self-filling syringe might get blocked by flesh in its needle.

Dr. A. FEILING said that the wheel goniometer was a useful and fascinating instrument for measuring the mobility of joints, but did not think that Kernig's sign was of much value in meningitis.

Dr. EVE (in reply to the President) said that the spring of the sensation tester should be strengthened, as suggested. He also said that although the President's great experience of cisterna puncture might enable him to dispense with these aids, most people would find them a safeguard to their patients and a relief to their own anxiety; no precaution should be omitted. He admitted that his figure-ratios for predicting depth were provisional and needed confirmation. But in the case of the fifteen subjects punctured, the maximum error was only 0·7 cm. This was half or quarter of the depth of the cisterna (1·5 to 3·0 cm.). Thus an ample insurance against puncturing the medulla seemed to be provided. He (Dr. Eve) did not stress the use of the self-filling syringe; it was an extra precaution and had not blocked itself so far. It was useful also for exploring chests and for ampoules. The aiming device worked well and obviated groping and uncertainty.

In reply to Dr. Feiling's reference to the failure of Kernig's sign in meningitis, Dr. Eve said that on the contrary, he agreed with an eminent neurologist who had found it the most useful sign in that disease. He (Dr. Eve) had discovered that its utility was much enhanced when forcible flexion was eliminated and the angle recorded daily.

Section of Odontology.

President—Mr. J. B. Parfitt, L.R.C.P.Lond., M.R.C.S.Eng.

The Relationship of Dental Infection to Diseases of the Skin.

By H. W. Barber, M.A., M.B., B.C.Cantab., F.R.C.P.Lond.

(Physician in Charge of the Skin Department, Guy's Hospital.)

Dental surgeons and dermatologists possess this advantage in common that they can actually see the parts of the body which they are called upon to treat, but they also share, with the throat, nose and ear surgeon a great responsibility both towards the patient and his medical adviser. That responsibility rests on the fact that in the majority of cases the earliest signs of ill-health are to be read in the skin and accessible mucous membranes.

Apart from the hereditary transmission of actual disease, such as syphilis, or of tendencies to disease, namely, susceptibility to infections like tuberculosis, and certain metabolic errors or idiosyncrasies—and this hereditary factor is of the greatest importance—the health of an individual is almost entirely dependent on what he eats and drinks, on physical hygiene, including muscular exercise, fresh air and sunlight, suitable clothing, etc., and on infection. With regard to infection, we have to consider that due to certain common micro-organisms, which we have always with us, and that due to more specialized ones, and conveyed to us by indirect or direct contact with another infected person, or by the intermediary of animal or vegetable hosts. It is with the former group of organisms—the common ones, which may be regarded almost as the natural flora of our skin and mucous membranes—that we are concerned to-day. Although the species of organisms found commonly in the skin differ from those present in mucous membranes, and although different species are found according to the part of the skin or the particular mucous membrane examined, nevertheless the primary factors which enable these common organisms to take on active growth, to increase in virulence, and to invade their host's tissues with pathological results, are probably roughly the same, both in skin and mucous membrane.

As regards the skin, Sabouraud [1] long ago pointed out that, although innumerable micro-organisms may be present on the surface, they exist, except in moist folds, such as between the toes, as isolated units, and are not in a state of active growth. On the other hand, as Whitfield [2] suggests, when an organism is found in a state of active growth upon the skin, it is exerting its noxious effect, if it has any. The same is true for the mucous membranes: those of the mouth, naso-pharynx, and intestines swarm with a great variety of organisms, but in a perfectly healthy person they are saprophytic and not parasitic.

It is clear, therefore, that in dealing with the question of actual infection with these common saprophytic, but potentially parasitic bacteria, we have to consider first their active growth in the host's tissues, and secondly, the immediate and

remote results of such active infection. The first part of the question is clearly biochemical or metabolic in nature: what, in fact, are the changes that take place in the host, rendering him a suitable culture medium for organisms which he normally harbours only as saprophytes? The second part of the question is concerned with the local pathological changes produced by bacterial invasion, and with the possible systemic effects that may arise from it in parts of the body remote from the original site of invasion; in other words, *focal sepsis* and its results.

In our consideration of the relationship of dental infection to diseases of the skin, the problem naturally divides itself into two stages: (1) The coincident surface infection of the skin and of the mucous membranes of the mouth and naso-pharynx, and the factors responsible. (2) The secondary effects of such infection upon other organs in the body, the skin itself being the organ with which we are chiefly now concerned.

The Coincident Surface Infection of the Skin and Mucous Membranes.

Ever since the war, I have been interested in this stage of our problem, namely, the susceptibility of some persons to infection of the skin and of the oral and naso-pharyngeal mucous membranes with certain common organisms, and the immunity of others. The conclusions I reached at that time (1917) [3] remain substantially the same, although they have been modified and extended. At the present time I am engaged anew, in conjunction with a biochemical colleague, in a systematic research upon the whole problem, which will, we hope, clear up several obscure points and provide scientific proof of much that is at present hypothetical. The subject is one of immense importance for many reasons. Thus the various organisms responsible for these surface infections, as has been said, are common saprophytes, and may be regarded, perhaps with some reservations, as normal inhabitants of the skin and mucous membranes. The conditions which predispose to the transformation of these harmless saprophytes into virulent and potentially dangerous parasites are obviously biochemical in nature, and dependent upon a change in the metabolism of the host. It is not merely a question of lowered immunity to one specific organism; it is a change which enables a number of organisms, of widely different characters, to grow actively and simultaneously at their host's expense and to produce a variety of morbid lesions. In other words, the clue to the problem is the chemistry of the soil, a factor that has been sadly neglected until recent years in the study of infective processes, in spite of the clear teaching of horticultural science.

A moment's thought will make one realize that the solution of this problem, if it could be universally applied, would have an incalculable effect on the standard of health among all classes. For by far the greatest number of chronic diseases, and many acute ones, depend directly or indirectly on the morbid activities of ubiquitous micro-organisms belonging to these common species, particularly the unstable and heterogeneous group of streptococci. If one could abolish dental caries, gingivitis, pyorrhœa, chronic naso-pharyngeal and nasal sinus infection, how many diseases and how much chronic ill-health would cease to exist? And, as regards my own speciality, were infections due to the common pyogenic cocci overcome, as all dermatologists will agree, a large part of our hospital and private practices would disappear. It is true that virulent strains of these common catarrhal —to use a word that is equally applicable to the skin as to the mucous membranes—

organisms may be conveyed from an infected to a healthy person, and cause an acute disease, such as impetigo contagiosa or follicular tonsillitis, and from this a subsequent chronic infection may ensue with secondary complications. But, on the whole, it is more usual for chronic infection of the skin and mucous membranes to begin insidiously with organisms of low virulence, and for the virulence to increase as the organisms multiply and the host's resistance diminishes, or for different species of greater toxicity to profit from the initial tissue-damages, and to become secondary invaders.

Let us, therefore, consider the factors which most commonly predispose to the active growth of the various catarrhal organisms of the skin and mucous membranes. I have already said that it is in the skin and accessible mucous membranes that the earliest signs of departure from perfect health are to be read in the majority of cases. Unfortunately, under the conditions in which we live, these signs are so common as to be neglected or even considered as normal. As regards the skin, the most frequent symptom of unfitness is the excessive and morbid secretion of the sebaceous glands, which we call *seborrhœa*, and which renders the skin a suitable soil for the growth of at least three micro-organisms, viz., the wrongly-called "*bottle-bacillus*," probably a yeast, which grows in the horny layer; the *micro-bacillus of acne*, which flourishes in myriads in the pilo-sebaceous follicles; and various strains of *staphylococci*. There is no longer any doubt, I think (although much biochemical research remains to be done on this question) that seborrhœa—or, as I prefer to call it, the *seborrhœic state*—is an index of faulty metabolism, there being an excessive formation of fatty acids, e.g., butyric and lactic, which remain unoxidized and are excreted through the skin. This excretion takes place through the sebaceous glands; the chemical composition of the sebum is probably altered (although to my knowledge this has never been demonstrated), through the sweat glands, the sweat of seborrhœic persons being often strongly acid even in situations, such as the palms and soles, where no sebaceous glands exist. The secretion is also probably effected through the epidermis as a whole, for, as Darier points out, there is a modification in the horny layer of the seborrhœic skin as regards its fatty content. It is presumably this chemical alteration in the direction of increased acidity that favours the growth in the skin of the micro-organisms referred to.

The chief factors in the ætiology of the seborrhœic state I believe to be the following:—

(1) *Heredity.*—A hereditary predisposition would seem to be certain; i.e., the child of seborrhœic parentage appears more liable to develop the seborrhœic state and its complications, given the other necessary predisposing factors, than one of non-seborrhœic parents.

(2) *Diet.*—This is, in my experience, by far the most important factor of all, or, at any rate, the correlation of diet and physical exercise. It is self-evident that, given a normal individual, his health must necessarily depend largely on those factors which in combination will make him a healthy animal, and, of these, what he eats is surely of paramount importance. I am glad to admit that it is to members of the dental profession that we owe some of the most logical and scientific pronouncements on dietetics. With the writings of Dr. Sim Wallace [4] and Professor H. P. Pickerill [5] I almost completely agree, and from my own observations on the ætiology, prevention, and treatment of the seborrhœic state, I long ago arrived at conclusions almost identical with theirs. In other words, as regards surface infection of the oral mucous membrane and of the skin, the dietetic factor is the same. The chapter on "The Dietaries of Modern Civilization: Their Errors and Correction" in Professor Pickerill's book is one of the best summaries of this question that I have ever read, and I would put his book into the hands of every medical

student before he begins work in the medical wards. It is, of course, the unnatural, *soft*, insipid, easily-fermentable starchy foods and concentrated sweetstuffs that are largely responsible for dental caries and oral sepsis, whereas natural sapid foods, which require thorough mastication and which excite a copious flow of strongly alkaline saliva, afford a better protection than the most assiduous use of antiseptic mouth-washes and tooth-pastes. Moreover, foods which may be regarded as salivary depressants are, practically speaking, those that excite little or no gastric, pancreatic or biliary secretion, and it is precisely these foodstuffs, which predispose to dental caries and oral sepsis, that in my opinion are concerned in the causation of the seborrhœic state. At my request several medical men, who have consulted me, have experimented upon themselves, and all are agreed that by taking an excess of carbohydrate—particularly of *soft* starchy food and concentrated sweetstuffs—they can immediately increase the greasiness of their skin, and bring about an exacerbation of the secondary infection with the seborrhœic organisms for which they sought my advice. Certain fats, as might be expected, are also harmful, particularly, as Kenneth Wills has insisted, pig-fat, and some kinds of cheese.

To sum up, the type of dietary that favours the development of the seborrhœic state is, first, one that contains an excess of carbohydrate, particularly a diet of insipid, soft, farinaceous food, and of unnatural, concentrated sweetstuffs—food, in fact, which either has little or no power to excite the flow of saliva and the gastric and pancreatic secretions, or which is easily fermented by acid-producing bacteria with the formation of acids of the fatty-acid type and, secondly, one that is lacking in natural fresh vegetables and fruit which are rich in mineral salts and vitamins. The same dietary defects are largely responsible for the active growth of micro-organisms in the mouth.

(3) It follows from the above considerations that all conditions which favour delayed digestion and incomplete oxidation of carbohydrate—and to a less extent probably of fats—and bacterial fermentation of the former, will predispose to the development of the seborrhœic state. Insufficient muscular exercise, lack of fresh air, over-heated rooms, and excessive clothing, are therefore important factors, as also is a heavy infection of the alimentary tract with acid-producing organisms. Apart from a suitable dietary, the best preventive of the seborrhœic state is abundant physical exercise in fresh, cool air, which should have access to the skin of the whole body through light porous clothing. Several of my seborrhœic patients have told me that they can eat with impunity, when taking plenty of out-of-door exercise on holiday, foods which in their ordinary sedentary life would intensify their seborrhœic manifestations.

(4) *Insufficient Intake of Fluids.*—If, as I contend, the seborrhœic state depends on the accumulation and imperfect excretion of acid waste products, an insufficient intake of fluid may clearly be another important factor; women are the chief offenders in this respect. In most chronic seborrhœics, one finds an intensely acid early morning urine with high specific gravity, and frequently there is no alkaline tide, the pH of the tide urine being often the same or even higher than that of the resting specimen.

(5) *Influence of Sexual Evolution.*—Most text-books, in discussing the ætiology of the seborrhœic disorders, emphasize the influence of puberty, of pregnancy, and of the menopause. Moreover, Sabouraud [6] has clearly pointed out the relationship that undoubtedly exists between the sexual glands and the pilosebaceous system. The sebaceous glands undergo increased development and become more active at puberty, at which time, of course, seborrhœa and acne vulgaris are likely to appear: but this is no justification for regarding them as the natural penalty for puberty, and for comforting the parents with the assurance that the child will "grow out of it."

Such an attitude of resignation may lead to permanent disfigurement and cause much misery. Acne vulgaris, acne rosacea, and other seborrhœic infections usually become worse just before the menstrual periods, and rosacea at the menopause. The influence of pregnancy varies: during it, seborrhœic manifestations may entirely disappear, but, on the other hand, may increase in intensity. These facts, however, merely emphasize the rôle played by the endocrine glands in influencing the general metabolism of the body. They do not affect the view already outlined as to the chief causative factors in the seborrhœic state.

What I have said concerning this seborrhœic state applies equally to the skin and to the mucous membranes of the mouth and naso-pharynx. Ever since the war, when my attention was first directed to the prevalence and probable ætiology of seborrhœic disorders, I have noted the state of the teeth and of the mucous membranes of the mouth and naso-pharynx in seborrhœic and non-seborrhœic subjects. My conclusions are: (*a*) That seborrhœics are liable to recurrent or chronic catarrhal infection of the naso-pharynx comparable to the "catarrhal" infection of the skin, the only distinction being the different species of organisms responsible. The mucous membranes of the intestines, too, are also apparently often affected, and balanitis and vulvitis are not uncommon. (*b*) That seborrhœic persons are prone to dental decay and to develop gingivitis and pyorrhœa alveolaris; but here it must be noted a good deal depends on the age at which the seborrhœic state supervenes. In some it begins in early years, and in them the teeth decay early, and gingivitis and pyorrhœa are well-established before adult life. In others, who spend their youth in active out-door pursuits, the teeth and oral mucous membrane may be in excellent condition, but, when they take to a sedentary life, without altering the amount and quality of their diet, seborrhœic symptoms appear and with them dental decay and oral infection begin. It is very rare to see good teeth and healthy gums in a bad seborrhœic of many years' standing—as a rule one either finds a complete set of false teeth, or two plates hanging on to a few doubtful teeth with infected gums around them. I do not wish to infer that persons with gingivitis and pyorrhœa are always seborrhœic; there are other conditions, such as acute general infections, deficiency diseases, such as scurvy, metallic poisoning, etc., which may act as predisposing causes. (*c*) That the persistence of the seborrhœic state leads to a drain on the available calcium in the body, owing to the necessity of neutralizing the excess of acid produced, so that symptoms of calcium deficiency may supervene. It has been found, of course, that dental decay takes place owing to the local action of acid produced in the mouth by the effect of bacteria on fermentable carbohydrate (Miller 1890, McIntosh, James, and Lazarus-Barlow 1922), but it is possible that such calcium deficiency may be a factor.

If these views are substantially correct, it is obvious that legislation is urgently needed to alter the economic aspect of foodstuffs. The policy of provision of cheap bread, sweets, cakes, sugar, jam and tea for the people, is fundamentally and criminally wrong. There is irony in the fact that it is precisely the most harmful foods that have been made the cheapest, whereas fresh fruits and green vegetables are among the most expensive, and beyond the means of a large section of the community.

The Secondary Effects of Infection of the Skin and Mucous Membranes upon the Organs of the Body.

Coming now to the second stage of our problem, we have seen that in the seborrhœic state the skin becomes a suitable soil for the growth of organisms, such as staphylococci, which are present on normal skin as inactive saprophytes, and that

44 Barber: *Relationship of Dental Infection to Diseases of the Skin*

the mucous membranes of the mouth and naso-pharynx harbour numerous bacteria, the most important varieties of which belong to the streptococcal group. Once such organisms begin to grow actively in the tissues, their virulence, of course, increases, and all the complex mechanisms of tissue-invasion and toxin-production on the one hand, and tissue-defence and antibody-production on the other, come into play. We have then to consider not only the local effects of microbial invasion on the skin and mucous membranes, but also the remote results of such local infection on other parts of the body, either owing to absorption of toxins or to the actual escape into the lymphatics and blood-stream of micro-organisms. In other words, we have a focal infection, whether it be tuberculous, diphtheritic, streptococcal, staphylococcal, or what not, and we witness the general systemic effects that result from the failure of the local tissue-defence to overcome such infection.

I would emphasize one remarkable fact, namely, that the doctrine—to use a non-committal term—of chronic streptococcal infection, which in recent years has more and more influenced medical thought and teaching in this country and in America, is practically unrecognized on the Continent and elsewhere. It is true that in France works have appeared on pyorrhœa and the various conditions of ill-health that may result from it, but the general pathology of focal streptococcal infection is, on the whole, either not understood or wilfully ignored except in English-speaking countries. As a proof of this, one has only to look up the papers on focal infection in the *Quarterly Cumulative Index to Current Medical Literature* for the past few years; with hardly a single exception every one has been published either in American or English journals. By way of contrast, a similar inquiry into the literature of tuberculosis yields a large number of references to papers in Continental and Scandinavian publications. With regard to my own subject, I may say that Continental writers—particularly the French—appear to be obsessed with two infections, viz., tuberculosis and syphilis; focal tuberculous infection, with its immediate and remote effects, is fully understood and appreciated, but focal infection with pathogenic streptococci, which is strictly comparable, and in my experience very much commoner, is completely ignored.

Although it is often forgotten, the skin itself may be an important primary focus of infection, and from such a primary focus lesions remote from it may result, either in the skin or in other organs. For example: (1) a chronic patch of infective dermatitis on a varicose leg may, if sensitization takes place, cause a widespread symmetrical eruption of infective eczematoid dermatitis on other parts of the body; (2) from a patch of ringworm on the scalp or elsewhere, dissemination of the actual fungus or of its toxins through the blood-stream may occur with the production of various types of eruption, corresponding morphologically to those produced by bacteria such as the tubercle bacillus, streptococci, etc., with enlargement of the lymphatic glands and spleen; (3) from boils or carbuncles staphylococci may be carried to a bone and set up an osteomyelitis, and I have known an extensive streptococcal ecthyma apparently cause acute nephritis. The likelihood, however, of an infective condition of the skin causing general toxæmic effects and bacteriæmia is certainly much less than in the case of the mucous membranes, doubtless owing to the greater protection afforded by the epidermis and its lesser vascularity. For instance, the profound systemic disturbance produced by a streptococcal tonsillitis and naso-pharyngitis, may be compared with the usually mild general symptoms associated with even a severe impetigo contagiosa.

Those who profess to be sceptical of the so-called theory of focal infection are merely betraying their own ignorance, and their inability to reason intelligently from such knowledge as they may possess of general medicine. For almost every pathogenic organism provides an illustration of the results of a focal infection; the primary

chancre of syphilis is the original focus from which practically every organ in the body may be affected; a tuberculous lymphatic gland may be the original source of infection in tuberculosis of the meninges, peritoneum, pleura, lungs, kidney, bones, skin and so on; a diphtheritic infection of the throat may cause neuritis, myocarditis and nephritis; a pneumococcal invasion of the lung may be complicated by arthritis, colitis, endocarditis and meningitis; and if these sceptics admit, as they must, that an acute streptococcal tonsillitis may lead to arthritis, nephritis, endocarditis and various erythematous rashes, why should they deny the protean pathological effects of a chronic streptococcal infection?

With regard to the teeth, one has to consider the effects of gingivitis, of pyorrhœa alveolaris, and of apical infection of dead teeth. The possible evil effects of constantly swallowing purulent material from the mouth and naso-pharynx must be admitted by all, but there are still sceptics among both the medical and dental professions, even in this country, as to the danger from so-called root abscesses. One cannot neglect the experimental work of Billings, Rosenow and many other workers in this field. Much of Rosenow's work reads as almost too good to be true, and many have failed to corroborate it. I can only say that his views as to the specific elective affinity of certain strains of streptococci for different tissues and organs—which specific character is rapidly lost on subculture—are largely confirmed by clinical experience. I confess that I find myself in complete agreement with Izod Bennett [7], who says:—

"Root abscess of the type here described is probably the most important of the dental conditions responsible for general disease, such as arthritis, and is almost invariably the result of treatment by dentists."

Dwight Chipman [8] was one of the first to suggest the wide influence of focal infection in diseases of the skin, and since then numerous papers have appeared on the subject in America and this country (Leslie Roberts [9], Barber [10], Cranston Low [11], Semon [12]).

The diseases of the skin which may be caused directly or indirectly by oral sepsis can be conveniently classified and considered as follows:—

(1) *Diseases due to direct spread of infection from the oral mucous membrane to the skin*, e.g., impetigo contagiosa, streptococcal fissure, *perlèche*, and relapsing streptococcal lymphangitis. Fissures at the angles of the mouth are not uncommonly seen in association with gingivitis and pyorrhœa. The *Streptococcus pyogenes longus* shows a predilection for muco-cutaneous junctions and the natural folds of the skin, where it is apt to give rise to painful fissures, from which an infective dermatitis spreads out on either side—giving rise to one form of so-called intertrigo. The condition known by the French as *perlèche* is merely a streptococcal fissure at a labial commissure with a dermatitis spreading out fanwise from it. Such fissures may be the starting point of ordinary impetigo contagiosa, and they may also be the portal of entry of the streptococcus into the lymphatics, and thus act as a focus of infection in relapsing lymphangitis, which in its turn may lead to permanent blocking of the lymphatics with swelling (one form of elephantiasis nostras). In such cases, however, an external fissure of this kind is not always present.

(2) *Diseases in which oral sepsis by its effect on the digestive processes, by setting up an infective gastritis and by causing a secondary infection of the intestinal tract with acid producing organisms, e.g., streptococci, thus favouring fermentation, may play a causative rôle.*—It is clear that a number of morbid conditions might be included under this heading, but I will refer to only one—rosacea, or, as it is sometimes

called, acne rosacea. There are usually two elements in rosacea, one, the flushing, at first temporary and later permanent, of the central parts of the face; the other a secondary infection of the rosaceous area, with the seborrhœic organisms. Rosaceous patients are always seborrhœic, but they do not necessarily suffer from oral sepsis. At the same time the constant swallowing of pus from an infected mouth, and a deficient number of teeth, may be important factors in some cases. In no cases is the effect of a proper diet so striking as in patients with rosacea, and it is interesting to note that Sabouraud, in one of his excellent volumes of "Entretiens dermatologiques," devotes a chapter to the cure of this and similar seborrhœic conditions by the omission of bread from the dietary.

(3) *Diseases in which the presence of an infective focus in the mouth lowers the resistance of the patient to pyogenic infections of the skin.*—As examples of these may be cited boils and certain forms of cutaneous streptococcal infections. In cases of chronic or recurring furunculosis the possibility of a focus of infection elsewhere should always be considered. I have seen several cases in which the treatment of sepsis in the mouth, or more commonly, perhaps, the removal of apically infected dead teeth, proved curative when other measures failed.

(4) *Diseases in which the epidermis becomes sensitized to toxins absorbed from the mouth and teeth.*—Strictly speaking there is only one disease that can be included under this heading, namely, eczema, which may be defined as an inflammatory reaction of the skin indicative of epidermal sensitization. The number of irritants capable of provoking an eczematous reaction in a susceptible person is legion. In some cases it is probable that the epidermis may become sensitized to bacterial toxins carried to it viâ the blood-stream from a septic focus, such as the mouth; even then, however, as Adamson and Whitfield have insisted, there is usually an external factor apparent, i.e., the eczema tends to occur in situations where physical agents, such as light, pressure, friction, etc., come into play. On the other hand, it is also probably true that in cases in which a known external irritant is the chief provoking cause of an eczematous dermatitis, oral and other sepsis may render the epidermis sensitive to the irritant in question.

(5) *Diseases which may be regarded as true anaphylactic reactions due to systemic sensitization to bacterial protein.*—Under this heading come urticaria, its subcutaneous form, angeioneurotic œdema, and certain mixed erythemato-urticarial eruptions, comparable to those provoked by foreign serums. As I pointed out some years ago [13], a considerable proportion—perhaps the majority—of chronic or recurring cases of urticaria or angeioneurotic œdema are due to bacterial sensitization, the usual infecting organism being a streptococcus; other organisms may be responsible, e.g., *Bacillus coli*, the gonococcus and a staphylococcus. One of the most striking examples of this kind was that published by Hurst [14]. Sometimes one meets with cases in which an erythemato-urticarial eruption co-exists or precedes an eczematous one, i.e., mixed systemic and epidermal sensitization. In an acute case of this kind, in which there was severe constitutional disturbance with pyrexia, apical infection of several dead teeth was shown beyond question to be the source of toxæmia.

(6) *Diseases in which the toxins from an infective focus act on the nervous or endocrine-sympathetic systems, the changes in the skin being secondary to such involvement.*—These include several very important conditions of great interest from their association with general medical diseases. As examples may be cited herpes zoster, alopecia areata, some forms of prurigo, vitiligo, scleroderma and Raynaud's disease. Herpes catarrhalis is, of course, due to a specific filtrable neurotropic virus, and certain bacterial infections predispose to its invasions, e.g., pneumonia, erysipelas, cerebro-spinal meningitis and the common cold. Herpes zoster, on the other hand, is

probably not a specific entity. In some cases it would appear to be due to invasion of one or more posterior root-ganglia with the virus of varicella, and in others to focal streptococcal infection with invasion of the ganglia through the blood-stream. The work of Rosenow is well known in this connexion.

With regard to alopecia areata, vitiligo, scleroderma and prurigo, these are conditions dependent on involvement of the sympathetic or endocrine-sympathetic system, and are not specific entities from an ætiological standpoint. The cause of such involvement is usually chronic infection, but not invariably. In all these diseases psychical factors, which play so important a part in disorders of the endocrine-sympathetic system, may be of importance, as they undoubtedly are in Graves' disease, in which alopecia areata, vitiligo and scleroderma so frequently occur. Most observers are agreed as to the importance of focal infection in alopecia areata, although, as Whitfield, Ravitch and others have pointed out, a few cases may be secondary to eyestrain or to reflex irritation from erupting wisdom teeth. I have had a remarkable case, to be published shortly, of long-standing scleroderma associated with hypothyroidism, rheumatoid arthritis, and Raynaud's disease, in which the patient recovered in dramatic fashion after removal of severely infected teeth and diseased alveolar bone. Dr. Graham Little [15] has published a similar case.

(7) Lastly we come to:—*Diseases due to inflammatory reactions produced in the skin as the result of bacterial embolism in the dermic or subcutaneous vessels, or by the action of bacterial toxins carried to the skin in the blood-stream.*—These include true erythema multiforme, erythema nodosum, the non-tuberculous form of erythema induratum, and lupus erythematosus. Of these I propose to deal only with erythema multiforme. This eruption, as you know, is often seen associated with, or alternating with attacks of acute rheumatism, and sometimes, like lupus erythematosus, in cases of rheumatoid arthritis. But the most interesting cases are those in which recurrent attacks take place at intervals, often over a period of many years, and in them the eruption occurs on the mucous membrane of the mouth, and sometimes of the vulva, as well as on the skin; moreover, it may be confined to the mucous membrane, its true nature being unrecognized owing to the absence of cutaneous manifestations.

In a long series of these recurrent cases, Eyre, Bulleid and myself have shown that the condition is due to chronic infection with and sensitization to a *Streptococcus longus*. The main foci of infection are usually the naso-pharynx and tonsils, or the teeth.

One case under my care was that of an old lady, who for ten years had recurrent outbreaks of bullous erythema multiforme on the arms and legs, which gradually became so frequent that she was hardly ever free from it. In this patient removal of some very septic teeth, with subsequent vaccination, led to a complete cure, and she has now been free from the disease for over three years.

Semon [16], Sutton [17] and others have described similar cases.

In conclusion, I would emphasize once more the paramount importance of studying the metabolism, the diet, the mode of life and surroundings of patients with chronic infective diseases. In such cases it is not merely a question of locating this or that focus of infection, of isolating the pathogenic organism responsible, and of vaccine therapy. We must try to discover the primary factors which lowered the patient's resistance to the infecting organism, and do everything possible to counteract these after the necessary treatment of infective foci has been carried out. Unfortunately it is rare to meet with the case in which the removal of an obvious septic focus leads to a dramatic and complete recovery; such cases do exist, but

more commonly secondary and inaccessible foci are present, and then treatment of the patient's general condition, as well as specific vaccine therapy and other methods of immunization, is necessary.

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Osteomyelitis of the Upper Jaw in Scarlet Fever.

By T. B. LAYTON, D.S.O., M.S.

THE first of the two specimens that I show you this evening is from a boy, now aged 6, who, some two years ago, was in the Eastern Fever Hospital suffering from scarlet fever, during the course of which œdema appeared below the left eye. This was followed by a swelling that appeared to be pus, and as there was a danger of the swelling bursting externally it was incised and some pus evacuated. As the condition did not clear up I was asked to see the child, and found that bare bone could be felt through the sinus. In addition there was a marked sublabial swelling on the same side. I thought the condition was one of suppuration in the maxillary sinus, and that it would clear if we opened it sublabially. This we did under light general anæsthesia, making a small snick with the knife and dilating the hole with sinus forceps, which passed up to the point near the orbital margin, where the abscess had been opened through the skin. This did not improve matters, beyond lessening the swelling of the cheek. Relatively large areas of bare bone could be felt and rocked with the probe, and so when the risk of spreading scarlet fever could reasonably be excluded I transferred the child to Guy's Hospital, where Mr. E. A. Scott very skilfully gave ether through an intratracheal tube. After making a sublabial incision the external wall of the maxilla proceeded to come away in small pieces and not as one sequestrum. When one or two such bits had been picked out I saw the fangless permanent lateral incisor lying like a pearl in a purple plush-like mass of granulations which filled its tooth-sac. This I picked out and immediately came on to another in a similar condition. In tracing the necrosed bone backwards I expected at any moment to open the maxillary sinus from which I thought the disease had arisen. To my surprise the outer surface of this membrane appeared convexly bulging into the wound, apparently normal. Needless to say I did not open it to assure myself there was no inflammation within, for to have done so would have

been inevitably to infect a cavity that up to that time might have been uninvolved. In the end I had found every tooth-sac infected and filled with granulations, with the fangless tooth lying in it. I had removed the whole alveolar margin on that side and a large part of the bony hard palate. To do this last I made an incision across the mucous membrane. This cut did not heal and dead bone could still be felt there. Through it a sequestrum subsequently peeped, and after waiting a long time to allow this to separate completely we removed it. The rest of the wound healed readily, but with that slowness which is characteristic of the surgical complications of scarlet fever.

The case proved, then, to be one of osteomyelitis of the maxilla arising in scarlet fever, and as it was not secondary to suppuration in the sinus, the question arose as to where the inflammation had arisen. I think it probable that it was secondary to a carious lateral incisor which was not there at the time of the main operation, but which was in place when first I saw the boy at the fever hospital.

On mentioning this case to Mr. Montagu Hopson I learnt he had never seen one like it. Dr. Goodall, in his long experience under the Metropolitan Asylums Board, believed he had seen two. Of one in the early years of this century he had no trace; but of the other he had kept the bits that came away and I am able to show them to you to-night. The case is not strictly analogous, for it was the lower jaw that was affected.

By the courtesy of Dr. Wilkins, the present Superintendent of the Eastern Fever Hospital, I can also give you the history of the patient Doris H——, who at the age of 3 was admitted on February 29, 1908, with scarlet fever of the septic type with muco-purulent rhinorrhœa, double cervical adenitis and laryngitis, with a slight stridor for which anti-diphtheric serum was given (8,000 units). On the next day the fauces showed ulceration, and this spread to the soft palate, so that it became perforated. She was fed through the nose. The laryngitis progressed and led on to bronchitis. On the tenth day after admission (twelfth of the disease) an incisor tooth fell out and other two were seen loose, the gums being ulcerated. There had been some swelling around one eye, for the notes say two days later that "the cellulitis of the eye had disappeared." More incisor teeth were lost a week later and by April 3 the sequestra that we see to-day had come away. The child was discharged from the hospital on April 14.

Dr. Goodall also drew my attention to a paper[1] in the reports of the Metropolitan Asylums Board. Dr. Mantell therein describes nineteen cases in a series of 12,230 cases of scarlet fever. In sixteen of these there was a common factor determining the necrosis, viz., mechanical injury. "The throat and mouth were, of necessity, frequently cleansed and food given at frequent intervals. The patient actively resisted this treatment." "The necrosis began in the height of the acute stage, close to the symphysis of the lower jaw." Seven of the sixteen patients died. In three only was the upper jaw involved and of these two recovered. It must be remembered that in these days syringing the throat was a common method of treatment, and for this to be done the gag was frequently needed in a recalcitrant child. Of the three other cases one was a case of general ulcerative stomatitis occurring in the fourth week and ending in death. The two others were associated with alveolar abscesses around a carious tooth and were therefore probably analogous to mine. In one the maxillary sinus was involved.

Our librarian has kindly investigated the recent bibliography of this condition and except for this paper can only find one passing reference[2] in which the writer speaks

[1] Mantell, H. F., "A Note on the Ætiology of Necrosis of the Jaw in Scarlet Fever," *M.A.B. Report*, 1897, p. 190.

[2] Babcock, W. Wayne, M.D., "Acute Osteomyelitis of the Jaw," *Journ. Amer. Med. Assoc.*, 1912, lix, p. 427.

50 Layton: *Osteomyelitis of the Upper Jaw in Scarlet Fever*

of the "well-recognized types of osteomyelitis following the exanthematous fevers, as small-pox, scarlet fever and measles," and a case[1] in which the artificial wall of the maxillary sinus was necrosed and perforated in a patient suffering from pan-sinusitis of one side during scarlet fever.

Dr. J. D. ROLLESTON said this communication which his colleague in the Metropolitan Asylums Board service had read was a very interesting one; interesting not only from the clinical point of view, but also from those of epidemiology and the history of medicine.

First, in regard to epidemiology, the character of scarlet fever had changed very much within the last half-century. It was now about fifty years since the Metropolitan Asylums Board hospitals came into existence, when the mortality from scarlet fever in this country was a little over 13 per cent., just about the figure it now stood at in Poland—in Warsaw the case mortality in 1926 was 13·5 per cent.—and ever since then there had been a gradual reduction in the mortality rate from it, until at the present time it stood at less than 1 per cent. At the date that Dr. Mantell wrote his paper, the mortality of scarlet fever was six times as high as now. Sixteen of his nineteen cases referred to by Mr. Layton were septic cases.

The question of the history of medicine came in in the following way: Sydenham said, when he described scarlet fever, that it was hardly worthy the name of a disease, and that the patient never died except through the doctor's excessive diligence. The deaths in Dr. Mantell's series were examples of the kind; the necrosis of the jaw being due to mechanical injury, caused by insertion of the spatula, the throat syringe, the finger, or the feeder.

Another point of view concerned the rarity of the condition. Though he (the speaker) had now been connected with fevers for over twenty-five years, he had seen very few cases of bone disease in scarlet fever, except in association with otological conditions; he never had seen a case of disease of the jaws in that relationship. In fact very little had been written about it in modern text-books, and he himself made no allusion to it when he wrote his book on infectious diseases. When a proof of Mr. Layton's contribution was sent to him (Dr. Rolleston), the first thing he did was to look at that mine of information, Misch's text-book of medicine and dentistry,[2] a copy of which he had brought to show Members. The writers in that book referred to several cases of scarlatinal necrosis of the jaw in out-of-the-way journals, which were not even in the library of the British Dental Association, though, by good luck, he found one of them in the library of this Society, with two illustrations of a case reported by Michel,[3] which he had thrown on the screen. The first view showed the facial aspect of the sequestrum of scarlet fever, the other the palatal aspect, with a very large sequestrum which was coming away.

This boy had not a very severe attack of scarlet fever, but there was this severe bone disease. The boy made a good recovery after the sequestrum was removed, fairly easily, and a prosthesis was made for him.

1 Lynah, H. L., "Osteomyelitis of the Sinuses of the Nose complicating Scarlet Fever," *Laryngoscope*, 1917, xxvii, pp. 176-180.

2 "Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde," 1923, i, p. 463.

3 *Correspondenz-Blatt f. Zahnärzte*, 1905-6, xxxiv, pp. 266-8.

Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

Clinical Meeting held at St. Bartholomew's Hospital.

Exophthalmic Goitre with Ocular Paresis.

By R. Foster Moore, O.B.E., F.R.C.S.

Patient, a woman, first seen by me in 1922. She then had severe exophthalmic goitre, for which part of the thyroid was removed. At that date she had pronounced proptosis, with paralysis of most of her external ocular muscles, but no involvement of the internal ones. She still has marked ocular palsy, which is little, if any, less than it was five years ago, and in consequence she has some difficulty in reading, as it is necessary to move her head from side to side so as to follow the lines of print on the page. Her general health is now very much better. I do not know where the lesion is situated in these cases, which I do not regard as common. There are no sensory symptoms, the internal branches are not affected, and I am disposed to believe that the lesion is a muscular rather than a nervous one.

Chronic Double External Ophthalmoplegia and Ptosis as an Isolated Lesion.

By Rosa Ford, M.B.

This condition is present in an otherwise perfectly healthy girl, aged 17. The first sign was noticed seven years ago, when she began to close one eye with her hand when looking at anything, probably on account of diplopia, which is still present. No cause for the onset is known. The condition caused her so little inconvenience that she did not seek advice until four years later, and then only because she was refused by the Civil Service examiners on account of her sight. She was then seen by Dr. Symonds, to whom I am indebted for a reference to Wilbrand and Saenger's description of similar cases.

There is now partial ptosis and almost complete ophthalmoplegia in both eyes. Each eye moves a very little vertically and horizontally from the straightforward position. The right eye is somewhat the more prominent of the two, and the left is a little divergent. Vision in right eye: $\frac{6}{6}$; in left, when fully corrected: $\frac{6}{9}$ partly. The fundi are normal, except that the outer halves of the discs are sligh tly pale.

The intra-ocular muscles have escaped, except that the pupil reaction to accommodation is very sluggish on both sides. The fields in this case, taken with a 1 mm. white object at 360 mm., show marked contraction, especially temporally, and both blind spots are enlarged. This suggests the possibility of a peripheral rather than a central lesion. The girl is a typist, and she says she does her work without difficulty.

The origin of the condition is doubtful. No cases of the kind have been examined post mortem. Wilbrand and Saenger, who collected thirty-two cases from the literature, confess that they are quite in the dark as to the cause, and I believe no cause has yet been agreed upon. The condition is not present in any other member

50 Ford—Davenport—Mayou—Scott

of this girl's family. Mr. McMullen and Mr. Hine, who in 1921 added two further cases, and with others collected from the literature, brought the number up to forty-seven, incline to the idea of a nuclear origin of the condition, the view being, apparently, that a congenital weakness of the nerve centres leads to premature decay. This theory would account for the facts that, though the condition is never present at birth, it is often found in several members of a family, that the cases present intermittency and variability and are progressive, though in some cases the whole course occupies more than twenty years, and that no other affection of the nervous system is present or develops in later life, even though some patients have lived to old age.

Bilateral Kerato-iridocyclitis.

By R. C. DAVENPORT, M.B.

THIS case is brought forward to show what I hope are the end-results of very severe inflammation of the anterior part of each eye in a child who has about her a condition strongly suggestive of tuberculosis and a bad family history. I am not prepared to say that the eye condition is tuberculous in origin; but she was found to have signs at each apex when examined at Brompton Hospital a year ago. At that time she had very intense inflammation in the lower half of each cornea; they were plastered with keratitis punctata, and the irides studded with nodules. She has been in the country since that time, and while she was there the condition subsided a good deal. The eyes are now quite quiet and the keratitis punctata has disappeared, but there is a little conglomerate exudate on the back of each cornea, interstitial nebulæ in each cornea, and dotted spots in each iris, these being, I think, remnants of the nodules. She has recovered extraordinarily well from what was a very severe, probably tuberculous, condition, and the general condition is greatly improved.

Degenerative Changes in the Cornea.

By M. S. MAYOU, F.R.C.S. (shown by R. C. DAVENPORT, M.B.).

THIS is Mr. Mayou's case, and I have his permission to show it: my case did not come. The patients are old ladies with atrophic changes in the back of each cornea, presumably colloid bodies are present in Descemet's membrane. They have each an atrophic iris behind the corneal atrophy, and each patient has fairly well marked lens opacities. Glycosuria is present.

Mr. M. S. MAYOU said that these thickenings of Descemet's membrane were well known pathologically. It was a long time since he had seen the case with the slit-lamp, but he thought that the endothelium was absent over most of the posterior surface of the cornea.

Unilateral Buphthalmos.

By RUPERT SCOTT, F.R.C.S.

PATIENT, a boy, aged 10.

The mother states that ever since birth the left eye has been larger and more prominent than the right.

The case is shown on account of the marked degree of ectropion uveæ in the affected eye, which gives the pupil the appearance of maximal dilatation.

Visual acuity, with correction, $\frac{6}{12}$; visual field shows slight concentric contraction; optic disc is slightly cupped.

When first I saw the patient, a few weeks ago, the tension of the affected eye was raised and arterial pulsation was observed in the arteries on the disc. The tension is now normal, and there is no arterial pulsation.

Mr. M. S. MAYOU said that this was an unusual case, as unilateral buphthalmos was a rare condition. When it occurred it was frequently associated with neurofibromatosis on the same side. On careful inspection of this boy it would be seen that his face was not quite normal, that it was thickened on the side of the buphthalmos, especially over the jaw, the fold on the top lid was obliterated; also he thought there was some proptosis on that side. There was marked ectropion of the uvea, which, he believed, had been present in some recorded cases. He (the speaker) regarded the case as a mild form of neurofibromatosis.

Left Macular Choroido-retinitis, with (?) Infiltration of Cloquet's Canal.

By RUPERT SCOTT, F.R.C.S.

THE patient, now aged 40, states that the affected eye was normal until the age of 19.

There is a large and circular deeply pigmented scar in the macular region of the left eye—a so-called macular coloboma. The retinal vessels running towards it stop at the edge. In one part of the area choroidal vessels can be seen. It is not ectatic.

In the anterior portion of the vitreous, and extending some little distance back, is a tubular-shaped opacity. There can be no doubt of its inflammatory nature; and it affords proof of the inflammatory origin of the scar at the macula.

A Case of (?) Coats' Disease.

By RUPERT SCOTT, F.R.C.S.

PATIENT, a boy, aged 16, was admitted into the hospital some weeks ago on account of periodic febrile attacks. The diagnosis is still in question. It is thought that he may be suffering from lymphadenoma.

I saw him four weeks ago, as he complained of defective vision of the left eye.

On examination, a zone of bright white patches of exudate was seen distributed in the deeper layers of the retina, around the macular region. Within this zone, a few hæmorrhages were seen and one small, very sclerosed vessel—a branch of the lower temporal artery.

I did not see the case again until to-day; the patches of exudate now extend farther afield.

The lower temporal artery presents a remarkable picture; for throughout its more peripheral course it shows numerous aneurysmal dilatations, which involve even some of the smaller branches. I am certain that this condition was not present at the time of my first examination.

Discussion.—Mr. WILLIAMSON NOBLE said he saw, at the Central London Ophthalmic Hospital, a case in which the anterior temporal artery was affected in the same way. The first time he saw the patient (a female) there were no fusiform dilatations, but on the second inspection they were evident. The interval between the two examinations was six weeks.

52 Scott—Penman—Hudson—Mann

There was in that case largely the same kind of appearance as in this, much œdema over the area involved. No cause for it could be found. His case was that of a girl, which was unusual, as most of the cases of the kind had been in boys.

Mr. R. FOSTER MOORE said that in one of the old *Transactions* there was a description of a case in which change occurred while the patient was under observation, i.e., in a very short time. Mr. Scott made it clear that the change in this case also occurred quite rapidly.

Cysts on the Optic Disc.

By G. G. PENMAN, F.R.C.S.

THIS man shows, on ophthalmoscopic examination, a transparent cyst coming from the centre of the left optic disc and overhanging the lower part of it. The vessels can be seen clearly through it.

On the right side there is something of the same sort, but this only appears as a fine membrane overhanging the lower part of the disc.

The vision is $\frac{6}{6}$, refraction + 0·5 D. sph. right and left, and there are no other abnormalities in the eyes.

These cysts appear to be connected with the hyaloid remnants. I am indebted to Mr. Foster Moore for permission to show the case.

Facial Cleft, Hare-lip, and Ectropion.

By A. C. HUDSON, F.R.C.S. (shown by C. H. ACKROYD, M.B.).

PATIENT, aged six months, came to the Royal London Ophthalmic Hospital two months ago, having had an operation for hare-lip.

The only operative treatment undertaken at this hospital has been a palliative one directed to obtaining a better approximation of the lids and indicated by the presence of keratitis from exposure. By this operation the lower lid was freed, the lid margins sutured and a Thiersch graft put into the raw surface beneath the lid.

The success of any plastic operation is at present complicated by the presence on the cheek of an orifice discharging pus, and it is considered that before any further operation is undertaken it will be necessary to obtain free drainage of the pus by partially reproducing the original deformity.

Mr. R. FOSTER MOORE said he did not think anything less than a well-planned tubular pedicled flap brought up from the chest could be expected to produce a satisfactory result in this case.

Exenteration of the Orbit for Perforating Melanotic Sarcoma.

By IDA C. MANN, F.R.C.S.

PATIENT, a tram conductor, aged 52, had been told in 1924 that the retina of his left eye was detached. He had taken no steps until, the eye becoming painful, he attended the Central London Ophthalmic Hospital in April, 1926, where I saw him.

He had an injected, painful glaucomatous eye with a melanotic sarcoma visible under the conjunctiva just behind the limbus on the temporal side. The orbit was exenterated and the cavity Thiersch-grafted about three weeks later. Sections of the eye showed a melanotic sarcoma of the ciliary body perforating the globe. The case is shown on account of the prosthesis, which is so good that the man has been allowed by the tramway company to resume work.

Demonstration of the Pecten in a Tame Owl.

By Ernest Clarke, C.V.O., F.R.C.S. (President).

I am exhibiting a tame owl in order to give the Members who have never seen a bird's eye with the ophthalmoscope an opportunity of seeing the pecten. It shows up very clearly in the owl as a deeply pigmented band arising from the optic disc, and curving forward into the vitreous, nearly approaching the lens. There are no retinal vessels, the pecten is supposed to take their place and supply nourishment to the retina. As it is a mass of thin-walled blood-vessels and pigment, it may exert a passive influence on the internal tension of the eye. The theory that it might act in accommodation by pushing forward the lens has been abandoned as the pecten contains no muscular structure.

Miss Ida Mann said that the reasons for supposing the function of the pecten to be nutritional were, first, that in birds there were no retinal vessels, and secondly, that the pecten was developed as an outgrowth of vessels from the hyaloid artery where it passed through the disc (in the same way as retinal vessels arose in mammals). Birds had an extremely small hyaloid system. At its proximal end the hyaloid artery passed through a small cone of cells (known as Bergmeister's papilla) derived from ectoderm and situated on the disc. This cone of cells became vascularized by branches from the hyaloid and, growing out into the vitreous, became the pecten. Microscopically, the pecten was composed of (mesodermal) capillaries with specialized walls and (ectodermal) pigment cells. There was no muscular tissue at all. The nutrition of the retina presumably took place by transudation of material from the pecten through the vitreous, and the whole mechanism appeared to be a specialization to allow of greater visual acuity than was possible when a layer of circulating blood-corpuscles was present in front of the percipient layer.

Deep Infiltration of Cornea, (?) in Descemet's Membrane.

By Leslie Paton, M.S. (shown by F. A. Williamson-Noble, F.R.C.S.).

This is a case of deep keratitis associated with iritis and cyclitis. The patient is a woman, aged 35, who says that fourteen months ago the left eye became very painful and inflamed. Within three weeks the right eye had also become affected. There

54 Paton: *Deep Infiltration of Cornea*

is much arthritis in shoulders, elbows, and knees. Several of her teeth have been removed on account of septic trouble, and she is now being investigated from the point of view of intestinal sepsis, tuberculosis, and syphilis. I think the opacity is not entirely confined to Descemet's membrane; I believe some of it to be new membrane which has grown up behind the cornea as a result of this chronic iridocyclitis. I have seen sections of an eye in which that has occurred.

Section of Otology.

President—Dr. DAN MCKENZIE.

Glosso-pharyngeal Facial Nerve Anastomosis.

By E. WATSON-WILLIAMS, M.C.

[ABSTRACT.]

WE owe this operation to Sir Charles Ballance [1], who was the first to perform it on a human subject. Two objections have been raised to it: (1) That the glosso-pharyngeal is a sensory nerve, and therefore unsuitable; and (2) this nerve is small, deeply placed, and only accessible with difficulty.

The first criticism is met by the results of experience; published cases show that voluntary movement is restored after this operation. The second point it is my purpose to refute.

Other methods of restoring movement to the paralysed face have not been entirely satisfactory. End-to-end anastomosis with the spinal accessory or hypoglossal entails interference with other muscles; should the facial result be disappointing the patient is worse off than before. If end-to-side anastomosis is employed, the difficulties of associated movements of the face with those of the arm or the tongue confront us. It is valuable to have a method utilizing a nerve which the patient can spare without inconvenience.

OPERATION.

With the head turned to the opposite side from that of the operation, and the neck extended, an incision is made from the front of the mastoid process to the great

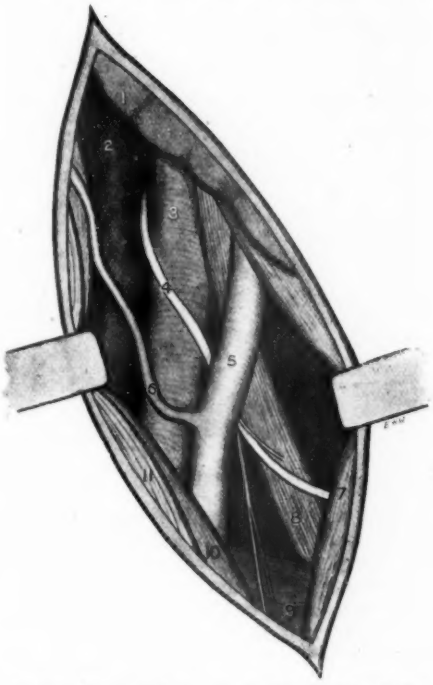


FIG. 1.—Exposure of glosso-pharyngeal nerve. 1. Gl. parotid. 2. Ven. jug. int. 3. Art. carot. int. 4. N. glosso-phar. 5. Art. carot. ext. 6. Art. auric. post. 7. Musc. stylo-hyoid. 8. M. stylo-pharyng. 9 M. constrict. med. 10. M. digastricus. 11. M. sterno-mast.

cornu of the hyoid. The anterior border of the sterno-mastoid muscle is defined, and retracted, the parotid gland is held forward, and the deep fascia is divided along the border of the muscle. From this point onward blunt dissection only is required to expose the nerve completely. The posterior belly of the digastric comes into view, and is followed down towards the hyoid bone. Here the tendon passes through that of the stylo-hyoid muscle. These two muscles are retracted, and blunt dissection between them reveals the external carotid artery, giving off the posterior auricular branch. Almost parallel and deep to the external carotid runs the internal carotid artery; separating them is the slender belly of the stylo-pharyngeus muscle,

FIG. 2.—Case 2. Note the contraction of right angle of mouth, previously paralysed.

along the posterior border of which runs the glosso-pharyngeal nerve, supplying the muscle, and crossing it just as the muscle is disappearing under the middle constrictor, to sweep forward beneath the stylo-hyoid ligament. Traced upward, the nerve is seen to emerge between the internal carotid artery in front, and the internal jugular vein behind. The fascia covering these need not be disturbed. Approached in this way the nerve is readily found and identified; it is the only nerve seen in the dissection. (The pharyngeal branch of the vagus lies behind and below, and is not exposed; no other nerves pass between the two carotids.) The facial nerve is now found in the upper part of the incision, curving round the styloid

process, and the anastomosis is made. Slender filaments from the glosso-pharyngeal nerve can be traced down on the middle constrictor. In the first of my operations I attempted to separate these and the branch to the stylo-pharyngeus muscle from the rest of the nerve, but after I had done so for some distance, the fine branches broke; I have since abandoned this practice as unnecessary. The glosso-pharyngeal nerve appears at this level to be nearly as large as the hypoglossal exposed in the usual position, a little lower (see fig. 1).

Illustrative Cases.

Case I.—Mrs. L. J., aged 56. Polypi removed from left ear in 1921, in 1923, and on January 30, 1926; "ear had run" all her life. February 1, 1926: Severe headache; ill for several days. On February 10: Had a rigor; was sent to me next day with diagnosis of meningitis. February 12: On examination: free purulent discharge from ear; fundus full of granulations; no œdema or redness, but definite tenderness over mastoid process; severe left side headache, no earache. She lay on her back, but was able to turn and lie in any position. Mixed spontaneous nystagmus to right in all positions of eyes; denied past or present giddiness; was confused, and her answers were delayed but rational. No paralyses; knee-jerks normal; plantar reflexes normal; Kernig's sign not definite, but head retraction quite definite. Temperature, 101·2° F.; pulse, 72: constipated. Dr. R. C. Clarke saw her, and considered meningitis definite. *Lumbar puncture:* Cerebro-spinal fluid, under considerable pressure, opalescent. Report by Dr. Fraser: "Many pus cells seen, no organisms"; no cultures made. After the puncture patient said she felt better. Completely deaf in left ear; caloric vestibular test negative. Now said that she had been giddy for several days, even falling down on left side.

Operation: Left radical mastoid. Antrum occupied and expanded by a putty-like mass of cholesteatoma; after removal of this, the facial nerve was seen lying on a bed of granulations: the foramen ovale open and exuding pus; promontory and labyrinth capsule necrotic. Complete labyrinthectomy was performed, with opening of the internal auditory meatus—blood and pus escaped under pressure. During this part of the operation the facial nerve was divided. Patient was delirious all night; next day fever had abated. Cerebro-spinal fluid was draining freely. On the fourth day the drainage of cerebro-spinal fluid ceased and there was recrudescence of grave symptoms; curetting the internal meatus restored the flow, and convalescence thereafter was uneventful.

September 15, 1926: There had been complete R.D. for seven months, with no change at all in flaccidity of the face. I performed anastomosis of glosso-pharyngeal nerve (less nerve to stylo-pharyngeus and branches to pharyngeal plexus) end-to-end with facial.

November 29, 1926: Epiphora less, and cheek less flabby; appearance unaltered.

January 17, 1927: Facial asymmetry obviously less, slight voluntary movement of angle of mouth. No return of faradic sensitivity.

February 5, 1927: Patient was shown at a meeting of this Section; some Members did not agree that there was movement of the mouth.

February 20, 1927: Report from massage department, Bristol Royal Infirmary: "Faradic response definite from depressor anguli oris, depressor labii inferioris, and adjacent part of orbicularis, none from other muscles."

The condition of the face continues to improve, and there is now no obvious asymmetry in repose. About a week after the operation the patient had for a few days a feeling of stickiness in swallowing, but no other inconvenience; there is no evidence of any paralysis following division of the glosso-pharyngeal nerve, but taste is impaired over the left half of the tongue.

Case II.—T. C., male, aged 36. Mastoid disease, 1916; complete paralysis before operation (right side). In September, 1926, patient came to me for treatment for epiphora. The cheek was completely flaccid and R.D. was found in all muscles of face.

November 9, 1926: I performed glosso-pharyngeal-facial anastomosis, using the whole of the former nerve. The facial nerve was apparently only a strip of fibrous tissue, and was found with great difficulty by tracing the posterior auricular branch from the artery.

February 8, 1927: Some change in appearance; visible movement of orbicularis palpebrarum; flabbiness unchanged, but there is a twitching sensation in cheek, especially at night.

62 Watson-Williams: *Glosso-pharyngeal-Facial Nerve Anastomosis*

March 1, 1927: Patient is able to whistle; movements of angle of mouth quite vigorous; faradic response obtained from depressor anguli oris. The eyelid is giving trouble, as a canthoplasty has been done and there is now trichosis. There is visible voluntary movement of occipitalis. The patient about a month ago had sensations when the cheek was galvanized, as of something pricking his throat; now he has this sensation intermittently, apart from treatment. No difficulty in swallowing; no change noticed in taste, which was impaired in right half of tongue before operation.

Discussion.—Sir CHARLES BALLANCE said that he had not invented the glosso-pharyngeal nerve operation; it had been suggested to him in 1895 by Sir Edward Sharpey-Schafer. He (Sir Charles) had rejected it because he thought the spinal accessory and hypoglossal nerves were more easily reached and would probably be more useful for the purpose. Moreover, both these were motor nerves, and it used to be considered important to use a motor nerve. The experiments which Mr. Colledge and he had carried out together showed clearly that there was no single nerve in the neck, including the cervical sympathetic, union with which to the paralysed facial nerve was not followed by recovery of faradic contractility in all the muscles of the face.

With regard to the glosso-pharyngeal nerve, the picture Mr. Watson-Williams projected on the screen represented the nerve as so large that it gave a false view of its surroundings. The glosso-pharyngeal, in monkeys and in man, was a very small, white nerve, much smaller than the vagus, and it ran a "crinkly" course across the muscles of the pharynx. The reason for this was that these muscles were always contracting, and therefore the nerve was compelled to lengthen and shorten, just in the same way as the facial artery did.

Mr. Watson-Williams had spoken of using the knife for cutting the deep cervical fascia along the anterior border of the sterno-mastoid, and then employing blunt dissection. The older surgeons, for instance, Sir Thomas Smith, one of the most skilful operators he (Sir Charles) had ever seen, never used blunt dissection, they used the knife and forceps; everything was cleanly cut.

This nerve was a very delicate one, very little blunt dissection should be done in these operations upon it, though sometimes, of course, it might be necessary.

In using the glosso-pharyngeal nerve it was important to proceed with the utmost gentleness, otherwise the best results were not likely to be obtained.

In the experiments which Mr. Colledge and himself had carried out together on baboons and other monkeys, they had obtained complete symmetry of the face within two and a half months, and a return of faradic contractility in all the muscles of the face within three months, and that seemed to be much earlier than in the cases recorded by Mr. Watson-Williams. Of course, in Mr. Watson-Williams' second case the nerve had been paralysed for ten years, and that fact made a considerable difference.

The exhibitor had referred to the spinal accessory and hypoglossal nerves. If the spinal accessory was chosen there was no method of using it without causing some permanent atrophy of the sterno-mastoid or trapezius, or both, thus destroying the symmetry of the neck, which, especially for a woman, was an important matter; and further, dissociated movement was not obtained when the hypoglossal nerve was used; there were associated movements of the face during eating and swallowing. He (Sir Charles) had shown a patient at a meeting of the Section three years ago in whom this condition was very evident. He had dined with her, and as she ate with relish an unpleasing distortion of the face occurred during the meal; and this was especially trying to him when he reflected that he had himself performed the operation.

After descendens noni—hypoglossal—anastomosis in the monkey, the eye on the side of the operation would wink while he was eating, almost closing, and the pinna would move up and down. When descendens noni—facial—anastomosis has been performed on human beings he (Sir Charles) believed these movements were scarcely noticeable.

One point he wished to emphasize was that in a case of facial palsy it was of supreme importance to keep up the angle of the mouth by means of a hook, otherwise it would droop, and before the anastomosis was complete the angle muscles might lengthen and so prevent a good result.

The exhibitor had stated that in one case he had found merely a piece of fibrous tissue with which to unite the nerve. If that had been actually so, he could never have succeeded

in getting a return of faradic contractility in the muscles of the face. In all his (the speaker's) own experiments he had been convinced, by microscopical evidence, that there was a certain amount of peripheral regeneration in a divided nerve, and many authorities to-day held the same view. This theory would explain why, after ten years' paralysis, Mr. Watson-Williams was able to obtain recovery in the face by anastomosis of the glosso-pharyngeal with the facial nerve.

The chief difficulty in making this anastomosis appeared to be the clean exposure of the facial nerve. The plan he (Sir Charles) had always adopted was to divide the tendon of the digastric and turn the posterior belly of that muscle backwards. Thus one arrived easily at the sterno-mastoid foramen, as the inner extremity of the digastric groove ended at the foramen, which was a most important landmark.

Mr. LIONEL COLLEDGE asked whether the picture of the area concerned had been obtained from a post-mortem specimen or at an operation. He (the speaker) did not understand how the nerve could be exposed in that way without dividing the digastric. He had never seen a large nerve trunk running down in the way indicated.

With regard to the use of the descendens noni in these operations, that nerve was exposed more easily than the glosso-pharyngeal. In spite of what Mr. Watson-Williams said, he (the speaker) considered exposure of the glosso-pharyngeal nerve very difficult and tedious. At a previous Meeting he had shown a case in which all the muscles had recovered their faradic contractility after anastomosis with the descendens noni.

In the monkey, the posterior auricular branch of the facial was very large. The monkey had large auricular muscles, and when the animal swallowed, the auricle moved up and down. This did not occur in the human being. After this operation in the human being the face movement during swallowing was so slight that there was no disfigurement.

Mr. F. BRAYSHAW GILHESPY said that in Sir Charles Ballance's book on the temporal bone, an operation performed by Mr. Sydenham was mentioned, namely, the implantation of silkworm-gut into the canal of the facial nerve, the result having been most successful.

Mr. WATSON-WILLIAMS (in reply) said that his patients had had thin necks, and he had not found any great difficulty in exposing the glosso-pharyngeal. He had drawn the diagram at the second operation. Perhaps he had been tempted to exaggerate the size of the glosso-pharyngeal nerve, as at the first operation he had found a much larger nerve than he had expected. He was also impressed by the way in which the nerve ran down the back of the stylo-pharyngeus and curled forward across, so that he was certain that this was the nerve for which he was looking.

He did not use any force in blunt dissection; force was not necessary. Until the nerve was exposed and identified he did not divide anything after passing the deep fascia. He was looking for a nerve which he had never before exposed in a patient, and, of course, he did not want to find it by the process of dividing it. It was a conspicuous object when one approached it in the way he had described. It was not necessary to divide the digastric. If one kept above that muscle the exposure of the nerve, at least in a thin neck, was easy and certain.

X-ray Skiagram by Dr. Gordon Thomson showing a normal Bony Auditory Canal in a Case exhibited at a previous Meeting, of a Boy, aged 4, with a Rudimentary Auricle.[1]

By H. J. BANKS-DAVIS, M.B., F.R.C.P.

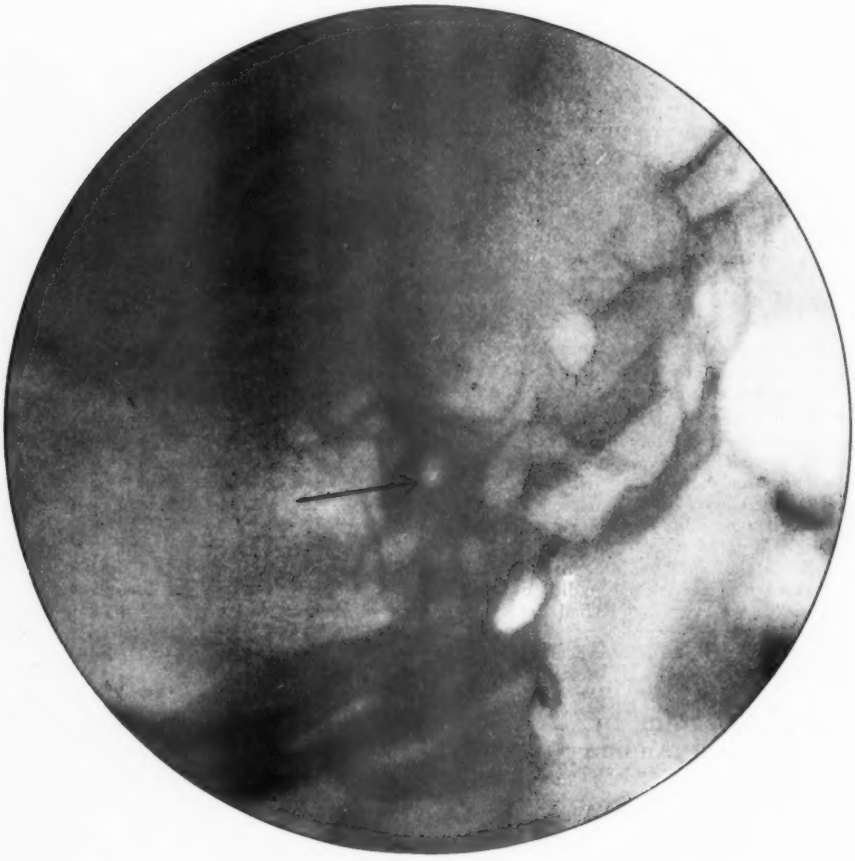
I SHOULD be glad of opinions as to whether any operation would be likely to improve the hearing. There were great difficulties in taking the skiagram, as the boy could not be kept steady.

Discussion.—Dr. DAN MCKENZIE (President) said that the presence of a bony auditory meatus in a case of rudimentary auricle was unusual. Did the clear space in the skiagram represent the bony meatus? If the boy had a bony meatus it would surely be skin-lined, and that would mean further trouble, because desquamation would be going on inside. As a rule, the external meatus was as rudimentary as the auricle in these cases.

[1] See *Proceedings*, xx (Sect. Otology), 25.

64 Banks-Davis: *Case of Rudimentary Auricle*

Mr. A. R. TWEEDIE said that he would hesitate to agree that the skiagram necessarily showed the external auditory meatus [Mr. BANKS-DAVIS: The term Dr. Thomson used was "normal auditory canal"], even if this latter term were intended to include the external auditory meatus and middle ear. But in any case the important point to be determined was the integrity of the inner ear. Was there any such case on record in which the hearing, even in a slight degree, had been restored by operation?



Dr. P. WATSON-WILLIAMS suggested that, in such cases as this, stereoscopic skiagrams would afford much more information than ordinary single plate ones, and would show up details in a very useful way, e.g., not only mastoid-cells, but even the bony labyrinth. When, as in the present case, the child could not be kept steady for the taking of the photograph, and the question at issue was important for the patient's welfare, it was justifiable to administer an anæsthetic.

Sir CHARLES BALLANCE agreed with Dr. Watson-Williams that a stereoscopic picture was the only kind which enabled an observer to see the external auditory meatus; and the surgeon should not be satisfied merely with the report of an X-ray expert, he should be able to see these things for himself. A long time ago he (the speaker) had operated in one of these cases on a person suffering from otorrhœa and he found a small antrum, and also a tympanum. No hearing had resulted from the operation, but the otitis was cured.

Mr. T. B. JOBSON said that X-rays might show the condition of the inner ear. Mr. Cathcart had shown to him (the speaker) a skiagram of a patient with otosclerosis, and he had been amazed at the clearness with which the inner ear showed up; all the canals, the vestibule and the cochlea could be seen, and a comparison easily made between the two sides.

Dr. DAN MCKENZIE (President) said that as the X-ray report was so definite, Mr. Banks-Davis might be justified in making an exploration. [Sir CHARLES BALLANCE: No.]

Mr. E. WATSON-WILLIAMS said that he had been watching an almost exactly similar case for four years, the child being still too young to be tested. All that he (the speaker) had been able to suggest in the way of treatment was a plastic operation later to remedy the deformity.

Mr. BANKS-DAVIS (in reply) said it had been suggested that if an external aperture were made there was a possibility of obtaining hearing. The general feeling of the Members seemed to be against operation. He believed that the boy had some hearing on that side.

[*Postscript.*—The radiograph shows the external auditory meatus *and* the internal auditory meatus. It was compared with a radiograph of the opposite (normal) mastoid region and did not differ from it. It is also similar to other radiographs of the normal mastoid region. A satisfactory antero-posterior view was not obtained as the child was very restless and an anæsthetic could not be administered at the time. I have taken stereoscopic films of the mastoid region on a dried skull at an angle at which this region is radiographed and find that the stereoscopic view only shows that the external meatus is not on the same plane as the internal meatus. It does not give a clear indication of the canal itself.—GORDON D. THOMSON.]

Case showing the Influence of Insulin upon Acute Suppuration of the Middle Ear in a Glycosuric Patient.

By DAN MCKENZIE, M.D.

THE patient was a man aged 80, the subject of glycosuria, who had been undergoing treatment by insulin from time to time.

During one of his periods of abstention from insulin, acute suppuration of one middle ear developed, and a paracentesis was performed. After a few days the membrane closed and the acute symptoms recurred. His age and ailments made posterior mastoid drainage unadvisable and a second paracentesis was therefore performed.

Following this, the second intervention, Dr. A. L. Sachs, his medical attendant, resumed the administration of insulin, beginning with ½ c.c. twice a day. With this small dose no effect on the ear discharge was noticed. Several days later the dose was increased to 0·75 c.c., and improvement at once became evident, the discharge drying up and the incision in the membrane healing in a few days.

Abscess of the Temporal Fossa secondary to Antro-meatal Fistula.

By Dan McKenzie, M.D.

The patient, a child aged 6, came to the Central Throat and Ear Hospital with signs of an abscess in the temporal region. Fluctuation was felt in front of the auricle above the zygoma, where the main mass of the abscess was centred, and the swelling extended as far forward as the orbit, the eyelids being œdematous. Over the mastoid the swelling and œdema were slight.

The illness, according to the mother, had begun only a few days before, and she is firmly convinced that the child had never had any discharge from the ear. No discharge, as a matter of fact, was visible in the meatus until the child was on the operating table, although the house surgeon had removed from the meatus a pale, flabby granulation or polypus.

Operation showed that the abscess had stripped up the pericranium of the squama; the bone of the zygoma was also bare.

The mastoid process was opened and emptied; it contained some pus and granulations. No zygomatic cells were discovered, and the origin of the abscess was not made clear until a probe, passed along the roof of the bony meatus, entered what seemed to be a fistula leading to the mastoid antrum.

Fistulous openings between antrum and meatus are, of course, common enough in cholesteatoma. But the bursting of an acute abscess of the antro-tympanum through its anterior or external bony wall, without discharging into the meatus but tracking outward to form a subperiosteal temporal abscess, seems to be rather rare.

The possibility of an antecedent primary cholesteatoma eroding the antro-meatal wall occurred to us, but no sign of any cholesteatoma was found.

Discussion.—Sir James Dundas-Grant said that a good description of that kind of abscess was given in Gruber's work on otology, translated by Edward Law. Gruber laid down the rule that if the bulging was at the level of the upper third of the auricle, the opening should be made through the meatus, i.e., through its roof. But if the bulging was below the upper third, the opening should be into the antrum. Sometimes the opening in the roof of the meatus was the key to the whole situation. He (the speaker) had had one or two cases which illustrated this extremely well. Rapid recovery had taken place after the abscess had been opened in the temporal region and a counter opening made through the meatal roof. In the President's case, apparently, the pus had tracked from the tympanum, possibly from the attic, and found its way up between the cartilaginous and the bony meatus.

Mr. Nicol Rankin said that another very similar case had come to the same hospital. The posterior meatal wall was necrosed, and the pus tracked up along the zygoma, forming a large subtemporal abscess. It was an acute case and there was no sign of cholesteatoma. The patient was a child who had never had any ear trouble until three weeks previously. When he (the speaker) operated he had found the mastoid antrum full of pus, the deepest part of the posterior meatal wall had been destroyed, and the pus had tracked along the zygoma and under the temporal muscle. No zygomatic cells were found.

Mr. A. R. Tweedie said that a possible solution was that this was an instance of acute otitis media with the usual extension beneath the periosteum of the posterior meatal wall, and that the probe had followed this track into the middle ear and thence through the enlarged aditus into the antrum.

Dr. D. McKenzie (in reply) said that he had not been sure where the probe was, and so he had enlarged the opening outwards along the meatal wall. When he found that the probe was against the bone on the far side, he concluded that it was in the antrum.

Section of Otology 67

Furunculosis (Right Ear) followed by Necrosis of the Anterior Bony Wall of the External Auditory Canal.

By NICOL RANKIN, M.B.

MALE, aged 44, seen in May, 1925, complaining of five days' earache.

On examination a large tender swelling was found in the right ear on the floor of the meatus. No pus found on incision. Pain continued. Ear filled with granulations: mastoid tenderness with swelling suggestive of a developing Bezold's abscess. Patient was admitted, mastoid opened up but found healthy. Meatus and canal curetted thoroughly. Granulations reported: "simple granulation tissue." Wassermann: negative.

Patient seen from time to time in out-patients' department till September, 1926, when he was readmitted to the ward suffering from pain, discharge, and stenosis of the meatus. A loose piece of bone could be felt which seemed to be part of the posterior wall of the external auditory canal. Mastoid re-opened and posterior wall found healthy. Fair-sized sequestrum then removed through the meatus. There was no relief from the symptoms and for the third time the mastoid wound was opened. This time the whole posterior wall was taken away and a large sequestrum, almost half an inch square, was removed; this proved to be the entire anterior wall of the external auditory canal.

Discussion.—Mr. T. B. LAYTON asked whether there was any trouble with movements of the jaw at any time.

Mr. RANKIN (in reply) said that the jaw movements were not impeded, though immediately after the operation they caused pain. The parotid gland could be seen pushed back into the meatus when the patient opened his mouth. He questioned whether he ought to have called it furunculosis. The condition might have been secondary to disease of the bone itself.

Large Extradural Abscess with Collapse of the Lateral Sinus and Compression of the Left Lobe of the Cerebellum.

By NICOL RANKIN, M.B.

W. W., MALE, aged 24. Admitted November, 1924. Carried into hospital on account of extreme giddiness. Intense headache, vomiting, profuse discharge from left ear and tenderness over left mastoid. Patient drowsy. Some spontaneous nystagmus to the right. No fistula sign (left). Well marked dysdiadokokinesis (homolateral), arm tonus reaction (homolateral). Patient had had two well-marked rigors.

Operation.—Radical mastoid. Cholesteatoma found and very large extradural abscess drained behind left lateral sinus. Sinus opened and found collapsed and empty, with healthy walls. Left internal jugular vein tied. Large suboccipital abscess opened and drained some time later. Patient discharged well about middle of December but readmitted on December 30, very ill. Left thoracic empyema discovered. This was treated at University College Hospital, and the patient made a complete recovery.

Discussion.—Mr. A. R. TWEEDIE asked if Mr. Rankin could say whether the symptoms were due to compression of the cerebellum or to labyrinthitis. Had the patient now a normal labyrinth on that side?

68 Brown: *Arterial Bruit causing Tinnitus*

Mr. RANKIN (in reply to Mr. Tweedie) said that he was not able to say whether there was now a normal labyrinth. The patient had been almost moribund; having had merely enough sense to make movements as directed. He (the speaker) thought the case would be interesting to see, following those of brain abscess shown at the meeting last month. Here he thought the cause of the symptoms was extradural. The case was shown also because of the condition of the lateral sinus. He had never before found the lateral sinus quite empty with healthy walls. He wondered whether it was due to the pressure of the abscess sweeping the blood up and down and emptying the sinus.

Dr. DAN MCKENZIE (President) asked whether there was any question of posterior basal meningitis or meningeal irritation in this case, apart from the mechanical pressure of the extradural abscess.

Mr. LIONEL COLLEDGE asked why the internal jugular vein had been tied if the walls of the sinuses were healthy.

Mr. RANKIN (in reply) said that the patient had had two definite rigors and was very ill, therefore he (the speaker) did not wish to omit any procedure which might aid recovery. He had opened the sinus not knowing what he would find; he had been surprised to find nothing!

Arterial Bruit causing Tinnitus.

By L. GRAHAM BROWN, F.R.C.S.

MRS. K., aged 48, has complained of various kinds of head noises, worse always in left ear, since September, 1926. She now definitely states she hears a sound like the "cawing of a rook" in the left ear. The noises are very distressing to her and cause much sleeplessness and mental anxiety. Her systolic blood-pressure is 150.

By induction one can actually hear this noise, which is synchronous with the pulse and can be definitely controlled by pressure over the left common carotid.

Mr. GRAHAM BROWN said that he had actually heard the so-called "cawing of a rook" in hospital, by placing his ear close to the patient's face, at the same time occluding the other ear. The noise was said to be preying on the patient's mind to such an extent that even the possibility of suicide had arisen, so that the question of treatment had become urgent. At his (the speaker's) suggestion the patient had worn during the night a broad band tied round the neck, to this was attached a small pad which pressed gently on the carotid. This had given her some relief, but he would be glad of suggestions as to further treatment.

Discussion.—Mr. W. H. JEWELL said that he had once shown a patient suffering from objective and subjective tinnitus which, however, ceased during sleep. After the removal of adenoids it had ceased altogether. He (Mr. Jewell) thought the patient could exercise some voluntary control over it by contracting the muscles. The tinnitus resembled the ticking of a watch varying from 60 to 70 beats a minute, and could easily be heard at a distance of one yard.

Mr. E. WATSON-WILLIAMS said that at the worst one could divide the eighth nerve. He remembered a case of agonizing tinnitus for which the cochlea was destroyed, with completely successful results.

Mr. H. TILLEY said that the operation mentioned by Mr. Watson-Williams had been performed for tinnitus so severe and continuous that suicide had been threatened by the patient. The operation was discussed in the old Otological Society, and one or two cases were recorded in which the auditory nerve had been divided within the skull, but the tinnitus had been in no degree relieved, so that a central factor was probably associated with the symptom.

Sir JAMES DUNDAS-GRANT said that Dr. Reik, an American aurist, had described an operation of his own at the Boston International Otological Congress. He had put a metal band round the carotid artery, and was able to graduate the degree of constriction exerted on the vessel. Had the effect of compression of the vertebral arteries, i.e., compression in the sub-occipital triangles, been tried in the present case? He (the speaker) thought this should be done in all cases of pulsating tinnitus. The pressure could be kept up by means of pads

put into the depressions, and a strap passed round the head. Treatment directed towards the blood-pressure, and the administration of bromides, might do good. In obstinate cases sometimes the anode of the galvanic current was beneficial.

Mr. H. J. BANKS-DAVIS said that the cases of tinnitus the most difficult to treat were those in which the noise increased when the patients were lying down. The best all-round treatment he had found was to give a large dose of hydrobromic acid, with nux vomica, and to place a blister behind the ear. He would not give bromide, which was a depressing drug. In many of these cases the blood-pressure was low, rather than high.

Mr. GRAHAM BROWN (in reply) said he had decided that the sound was an arterial, not a labyrinthine congestion tinnitus; for that reason treatment was not here a question of sedatives, but of some drastic measure for giving relief. He thought the bruit was caused by the blood passing through a dilated vessel, or through a narrowed bony orifice, or past an atheromatous patch. He would not suggest such a thing as tying the carotid artery, but he would be interested in trying Sir James Dundas-Grant's suggestion of mechanically compressing the vertebral arteries, though he did not see how this would be more beneficial than his (the speaker's) plan of pressing on the common carotid.

Frontal Abscess; no Headache; no Localizing Signs.

By E. BROUGHTON BARNES, F.R.C.S.Ed.

A. T., MALE, aged 11. Swelling of the forehead developed on the third day of a severe "cold." Was first seen on the twenty-third day, found to have necrosis of the left ethmoid and extensive osteomyelitis of the frontal bone. The necrotic bone was removed and the patient progressed. On the sixty-first day he vomited twice and was drowsy. He had internal strabismus of the left eye. The ophthalmic surgeon reported: "Bilateral papillœdema—central nebula, left." Intelligence was normal. No headache. No other signs discovered. Right frontal abscess containing about 1 oz. of pus (pure *Staphylococcus aureus*) found at depth of ½ in. The dura over both frontals had already been exposed; the right lobe was explored first as the granulations over it were slightly less healthy.

The patient developed hernia cerebri and died from basal meningitis exactly three months after the brain operation. No other abscess was found post mortem.

Giddiness persisting after Radical Mastoid Operation.

By T. B. LAYTON, D.S.O., M.S.

B. C. M., AGED 32. Discharge from ear began when patient was in the Army in 1918. He underwent two operations, the second—in 1921—a radical mastoid. The wound did not heal perfectly but a false membrane developed, under which there was always some purulent secretion. An attempt was made to dilate the perforation in this membrane with a laminaria tent, so as to secure drainage, and on several occasions portions were punched away under cocaine anæsthesia, but the membrane always grew again very quickly. Attacks of giddiness began about 1922, and after a worse one than usual, in March, 1925, the wound was re-opened, a small piece of bone removed, and a graft applied to the cavity. Healing was rapid except in one minute spot in which it occupied over a year. During convalescence patient had many attacks of giddiness with sensations of stiffness at the back of the neck. These became fewer from October, 1926, onwards but there was a severe attack on February 3, 1927, and another on February 14, 1927. Five days after the second attack a "tight feeling" developed behind the ear and he then discovered a small crack in the ear. By means of mirrors he can look into his own aural cavity.

70 Dundas-Grant: *Demonstration of Induction of Optical Nystagmus*

Discussion.—Mr. H. J. BANKS-DAVIS said that patients who had undergone a radical mastoid operation, and were considered to be cured, frequently complained of giddiness in cold weather. The reason being that cold air set up currents in the external canal and these produced vertigo.

Mr. A. R. TWEEDIE said that everyone who had attempted to repair, or to operate again on an "old mastoid" well knew the difficulties entailed. In the present case it was impossible to give an opinion without having had an opportunity of testing the labyrinth, but appearances suggested a localized patch of osteitis in the neighbourhood of the external horizontal canal, and presumably it was recurrent inflammation of this lesion which from time to time caused the trouble. The effect of a light application of the actual cautery might be tried.

Dr. DAN MCKENZIE (President) said that Mr. Tweedie's suggestion was a very likely explanation in many cases of vertigo which persisted in a similar way and in which there were no fistula symptoms.

Mr. LAYTON (in reply) said that the operative success in his case was largely due to the skin-graft cut and inserted into the wound by Mr. Gill-Carey. It was a distressing case. From time to time the patient was obliged to be a few days away from his work owing to these attacks of giddiness.

Demonstration of Induction of Optical Nystagmus with Simple Apparatus.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

THE apparatus consists of a strip of white paper about 5 ft. long and 8 in. broad (or high). On it are transverse vertical black stripes 2 in. wide, separated from each other by spaces of about 7 in. The paper is drawn from one side to another in front of the patient while he looks straightforwards at the black stripes. His eyes then enter into active nystagmic movements in a direction opposite to that in which the paper is moved.

The method has some clinical value, more perhaps for the neurologist than for the otologist. If a patient's nystagmus is of labyrinthine origin, the optical nystagmus thus induced will counteract it. If it does not counteract it, the nystagmus is probably of some origin other than labyrinthine. The roll can be easily carried in the aurist's or neurologist's bag.

Dr. DAN MCKENZIE (President) said that the apparatus was very convenient, and that another useful device of the exhibitor's was the cold-air apparatus for labyrinth testing; it was much more cleanly and convenient than cold water.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Mr. J. P. Lockhart-Mummery, F.R.C.S.

DISCUSSION ON COLOSTOMY.

Mr. W. Ernest Miles

said that with regard to the lumbar colostomy of pre-antiseptic days, when surgeons were afraid to approach the bowel intraperitoneally, the chief defect of the measure consisted in the fact that the stoma in the colon was lateral and did not stop the passage of fæces beyond it. It had one virtue—protrusion of the mucosa never occurred. Now that resection with anastomosis could be performed, the operation of colostomy was needed chiefly for inoperable carcinoma of the rectum or pelvic colon; lumbar colostomy had been gradually superseded by sigmoidostomy.

At first this was made in the middle of the pelvic loop, and an opening was made in the parietes large enough to put the hand in for investigation. The result was that the spur receded so that the opening became a lateral one with all the defects of the lumbar colostomy combined with considerable protrusion. Later, Mr. Harrison Cripps had pointed out that protrusion could be prevented by making the opening high up in the pelvic colon. Much controversy had followed, the advocates of the low opening said that it was necessary to preserve a natural reservoir which more or less controlled defæcation. They forgot that, whereas Nature provided a loop for control, she had also provided an efficient lock in the form of the sphincter. He (Mr. Miles) had no doubt that Mr. Harrison Cripps was right about the site of election. The essential feature of a colostomy should be that it effectively prevented the contents of the bowel from passing beyond the stoma into the loop below. For this the permanence of the spur was essential. If the mesentery were short, congenitally or as a result of carcinomatous infiltration, it was impossible to deliver a loop long enough to make a good spur, and directly the rod was removed the spur would recede on the slightest tension from within.

Some had thought that peristalsis had something to do with recession of the spur and had therefore divided the bowel completely, making a terminal colostomy. In cases for which colostomy was now performed, however, the distal portion could not be occluded because of discharges from the growth. His (Mr. Miles's) objection to this device was that division of the bowel created a weak spot between the two openings and sometimes large hernial sacs appeared between them. Another

modification was to make the colostomy opening through the outer border of the rectus muscle. This gave good support but could only be done where there was sufficient length of loop of the pelvic colon. It did not, in fact, matter what method was adopted so long as the colostomy was effective.

Mr. W. B. Gabriel.

I believe the operation of colostomy is being done now more commonly than ever before. The operations in which surgeons tried to avoid colostomy at all costs by preserving the sphincters or forming a sacral anus, are now no longer carried out. We regard a permanent colostomy as an essential part of any radical operation for a cancer of the rectum. It follows that the operation is done in the case of adults who, apart from their growths, are often healthy and active, and who may with good fortune expect many years of useful life after their operation. A colostomy, if well planned and properly managed, can be one of the most satisfactory operations, and it is not to be regarded as a crippling operation. From the point of view of function we expect it to be more than an unpleasant alternative to intestinal obstruction. Patients with colostomies should be able to take part in their usual social life and pursuits very much as before, and so far as manual work is concerned, we have records at St. Mark's of very numerous patients who have gone back to their work with colostomies, the men as wage-earners doing heavy work, the women doing their full housework and bringing up their families. Many of these patients have been inoperable cases who have experienced sufficient relief to enable them to take an active interest in affairs instead of remaining invalids to the end.

We know that the practice of different surgeons varies considerably in certain points, for instance, in the technique of the operation, in the management of cases, in the application of belts, etc. It is hoped that this discussion will be useful in the explanation to us of these different methods and in helping us to general improvement in the treatment of colostomy cases.

I propose to make my contribution to this discussion quite shortly under four heads, namely: (i) The common indications; (ii) the technique; (iii) the complications; and (iv) the management of colostomy. My remarks are based on the experience and practice of St. Mark's Hospital. I shall limit my remarks to colostomy of the pelvic colon, with the bowel in continuity.

(I) Indications.

(1) *Carcinoma of Rectum.*

(*a*) *Operable.*—A colostomy is now universally regarded as an essential part of a radical operation for cancer of the rectum; it will either be a terminal colostomy or one in continuity according to whether an abdomino-perineal or a perineal excision is done. The relative frequency of these two operations at St. Mark's for the years 1910-1926 inclusive is 55 to 187.

(*b*) *Inoperable.*—Nearly every inoperable cancer of the rectum is benefited by a colostomy if the operation is done in good time. If the operation is delayed until the patient is profoundly septic and anæmic, improvement and relief can hardly be looked for; for instance, if a patient is seen with a large fungating mass in the middle-third of the rectum with secondaries in the abdomen, flabby abdominal muscles and an advanced state of cachexia, very often such a case will proceed to its

termination under medical treatment with no more discomfort than would be present if a colostomy were performed.

The indications for colostomy in inoperable cancer of the rectum are well stated by Jacobson[1]:—

(1) Impending obstruction.
(2) Pain.
(3) Loss of control from involvement of sphincters.
(4) Profuse discharge and hæmorrhage.
(5) Multiple peri-anal fistulæ.
(6) Recto-vesical fistulæ.
(7) Cellulitis of buttock.
(8) Mass of growth outside the anus.

Generally it may be said that colostomy is required early for growths in the upper third of the rectum on account of liability to obstruction, and in the lower third for relief of pain due to involvement of sphincters. For a growth in the middle third (ampulla) a colostomy can usually be promised to give relief, somewhat variable in extent, from rectal discharge and bleeding, and by its enabling the surface of the growth to be kept clean and free from fæcal irritation there is less liability to the inflammatory complications enumerated above.

It cannot be denied that a definite proportion of cases occur in which relief is not obtained; this may be due sometimes to faulty technique (deficient spur) or to mismanagement, or to inability of the patient to accommodate himself to his new conditions.

(2) *Fibrous Stricture of the Rectum.*

A colostomy is required for strictures of the rectum which are too extensive or too high for dilatation, for cases in which dilatation causes great and continued pain or where dangerous reaction in temperature and increased discharge persistently follow dilatation.

(3) *Diverticulitis.*

This subject was discussed at the last Meeting of the Sub-Section,[2] and I need only mention here the pressing indications for colostomy, namely, acute diverticulitis with abscess formation or peritonitis, intestinal obstruction, and vesico-colic fistula. Colostomy in these cases may be temporary, pending cure of the condition by time and rest, or by surgical removal, but in many cases, even if the acute symptoms subside, it is advisable to leave the colostomy as a permanency.

(4) *Acute Spreading Ulceration.*

As a post-operative complication of hæmorrhoid operations, this used, so I am told, to occur quite commonly. Fortunately, it is now a very rare indication for colostomy and I know of there having only been one case at St. Mark's Hospital in recent years.

A case of piles was operated upon after the customary preparation and with the usual technique. A spreading ulceration set in, with pelvic cellulitis extending up into the left loin. A colostomy was done and the patient finally recovered.

(5) *Injuries of the Rectum.*

In recent injuries, such as severe lacerated gun-shot wounds of the rectum, especially if associated with fractures of the sacrum and pelvis, Hamilton Drummond[3] advocated colostomy as a useful adjunct to complete local drainage.

1 "Operations of Surgery," 7th ed., by R. P. Rowlands and P. Turner, 1927, 269.
2 *Proceedings*, 1927, xx (Sect. Surg., Sub-Sect. Proct.), 57-90.
3 *Proc. Roy. Soc. Med.*, 1919-20, xiii (Sect. Surg., Sub-Sect. Proct.), 26, 27.

In the treatment of old injuries to the rectum, when extensive plastic operations are required for the release or repair of injured sphincter muscles, a temporary colostomy is sometimes a very useful manœuvre for obtaining rapid healing at the anus.

(6) *Compression by Extra-rectal Tumours.*

Colostomy is advised for relief of obstruction due to pressure of pelvic chondromata and sarcomata upon the rectum. This must be a rare indication, but I have seen several examples of a more common condition—advanced carcinoma of the cervix uteri—in which the mass of growth had so compressed the rectum against the sacrum as to necessitate the establishment of a permanent colostomy.

(II) TECHNIQUE.

(1) *Incision.*

The most serviceable incision is a vertical one, 1½ in. to the left of the mid-line, which splits the fibres of the rectus muscle. The upper part of the incision should reach above the level of the umbilicus. If the deep epigastric vessels present, they are ligated at each end of the incision. The advantages of this incision are (1) it is possible, if required, to carry out a general exploration of the abdominal cavity through it; (2) it is less likely than any other to be followed by a ventral hernia; (3) the transverse colon is available if required. I am quite convinced that the rectus incision is far superior to an incision through the oblique muscles in regard to the lessened risk of ventral hernia. I have many times seen a rectus colostomy which has been established for years without the least sign of a bulge of the abdominal wall at the site of the colostomy, but I cannot recall having ever seen an old oblique colostomy which has not developed a hernia, sometimes of a very large size.

I have tried some of the "fancy" incisions designed to give a bridge of skin between the two openings of the colostomy, but there seems to be no particular merit in them. The simpler the method the better. The whole essence of the operation is that a good spur should be formed.

(2) *Method of Fixation of the Bowel.*

The glass rod remains the best method; it is better than the Ward stitch. The rod should be put through the pelvic mesocolon ½ in. to 1 in. from the edge of the bowel, and by means of blunt dissection over the end of the rod, as it is pushed through the mesocolon, injury to vessels should be avoided. A circle of rubber tubing prevents any possibility of the rod slipping out. It does not appear necessary to fix the bowel by any other stitch; retaining sutures put through the bowel wall must increase the liability to infection of the abdominal wall. If the glass rod alone is relied upon for holding the bowel it *must* be left in place for fourteen days at least; premature removal may lead to retraction of the bowel. In suturing the abdominal wall nothing but catgut should be used for buried sutures, and all sutures should be interrupted. The strength of the abdominal wall at the site of the colostomy depends on the rectus sheath, hence the peritoneum, with posterior sheath and then the anterior sheath of the rectum, should be approximated in two layers working from the lower end of the incision towards the bowel until the closure is just sufficiently snug; a finger slipped in along the bowel is the best guide. Skin is closed by interrupted silkworm-gut sutures and a few tension sutures passed through

the anterior sheath are advisable. It is better not to put rubber tubing on the tension sutures in view of the increased difficulty in cleaning the abdominal wall after the colostomy is opened. For dressing, dry gauze is placed underneath the rod on each side and wrapped closely round the bowel.

(3) *Difficulties.*

The most common difficulty in the actual operation is that of bringing the bowel to the surface, due to shortness of the pelvic mesocolon, or its loading with fat, to adhesions, or to the high situation and fixity of the growth. In most cases division of adhesions on the outer aspect of the bowel makes an enormous difference in freeing the bowel, and it is very important that this should be done thoroughly in order to avoid undue tension on the bowel by the glass rod. There are some cases in which a pelvic colostomy proves quite impossible, and in such an event it is a simple matter to extend the incision upwards and bring out the transverse colon. In very stout people any type of colostomy may be an operation of the greatest difficulty.

(4) *Complications.*

These may be immediate or remote.

(1) *Immediate.*—In our records at St. Mark's Hospital we have notes of 259 colostomies performed from 1910 to the end of 1926 for relief of inoperable cancer of the rectum. Excision cases are excluded from this list. The immediate mortality has been thirty-five, i.e., 13·5 per cent., the causes of death being the following:—

(*a*) *Heart failure, pulmonary complications and exhaustion*, which account for twenty deaths.

(*b*) *Peritonitis.*—The cause of death in four cases, one from extension of suppuration in the abdominal wall, one from slipping back of the colostomy owing to premature removal of the rod (second day), one from retraction of the bowel due to ulceration of the spur by the rod (eighth day), and one following resection of small intestine which was found adherent to the growth at the time that the colostomy was performed.

(*c*) *Prolapse of small intestine* by the side of the colostomy, accounting for three deaths. This accident may occur within a day or two of operation owing to inadequate closure of the abdominal wall, or it may supervene after seven to ten days, being due to sepsis and cutting through of sutures.

(*d*) *Intestinal obstruction.*—This proved fatal in one case; here a loop of small intestine became strangulated through the foramen between the colostomy and the abdominal wall, and the patient did not survive the second operation.

(*e*) *Obstruction.*—There is an indefinite group of four cases in which the patients remained distended after the colostomies were opened and wash-outs persistently failed to bring relief. Death occurred with continued obstruction, whether due to multiple obstruction from secondary deposits or some other cause is not clear.

(*f*) *Miscellaneous.*—Lastly, three patients died from miscellaneous causes—coma, renal failure and hæmorrhage from the proximal end of the colostomy.

(2) *Remote.* (*a*) *Stenosis.*—This may occur gradually, months or years after the colostomy has been performed; it is due to contraction of scar tissue, both round the colostomy and sometimes actually in the wall of the bowel. In my experience the narrowing is always at the level of the skin, and never deeper in the abdominal wall. To prevent it, I think it is wise not to suture skin too closely round the bowel. For a colostomy which is tending to become tight, the patient can usually prevent further narrowing by passing his little finger into each opening

every week or so, but in spite of this the narrowing sometimes progresses until a further operation is required to enlarge the orifice.

(*b*) *Retraction of the Spur.*—This is a very troublesome complication and one which must be actively treated to prevent the passage of fæces into the distal segment. Retraction occurs generally in cases in which difficulty has been experienced in bringing the bowel to the surface and is due obviously to tension on the bowel. It may also be due to trimming the colostomy too close to the skin, and lastly, it occurs sometimes for no apparent reason when at the operation the bowel presented easily and everything promised well. For treatment, the colostomy must be re-established by dissecting it out and re-fixing with a glass rod; this can sometimes be effected extraperitoneally, but usually it is best to incise peritoneum all round the colostomy and procure a really ample amount of bowel outside if possible.

(*c*) *Prolapse.*—This may occur from either the proximal or, rarely, the distal colostomy opening. It is due to an abnormally wide opening in the abdominal parietes together with violent or irregular peristalsis. As prolapse usually occurs from the upper end, one suggestion that has been made is at operation to pull the colon taut from above; this is what I myself do, but as a matter of fact I do not believe it makes any difference which part of the pelvic colon is brought out, provided the rectus sheath is sutured securely and snugly round it. For an established colostomy prolapse I think a well-fitting belt is the best remedy. I have never seen amputation of the prolapsed bowel performed, but presumably it might be undertaken.

(*d*) *Ventral Hernia.*—I do not think any technique will entirely do away with the development of a ventral hernia at the site of a colostomy. The question of the incision is a controversial one, but I would submit (1) that the left rectus incision gives a better abdominal wall than the left iliac incision, whether direct or of the "grid-iron" type; (2) that the wearing of colostomy belts with large cups seems definitely to promote the production of ventral hernia. I will refer to the question of belts again. Once a ventral hernia has developed the indication is to keep on supporting it; I should imagine that an operation for its radical cure in the presence of the colostomy would be a somewhat dubious one.

(*e*) *Extension of Carcinoma to the Abdominal Wall at the Site of the Colostomy.*—This complication is due to establishment of the colostomy too close to the primary growth. In such a case, if an adequate clear length of pelvic colon cannot be obtained above the growth it is better to perform a transverse colostomy.

(III) Management.

(1) *Opening the Colostomy.*

This is not done until forty-eight hours after operation unless extreme distension renders earlier opening unavoidable. Sometimes by giving small turpentine enemas from the second day onwards together with liquid paraffin by the mouth, opening of the colostomy can be postponed for as long as the seventh to twelfth day after operation. This is a great advantage for the healing of the incision. A colostomy should be opened with a Paquelin cautery in the transverse axis of the bowel, that is, in the line of the rod; the superficial half of the circumference only to be incised.

(2) *Trimming.*

If the amount of bowel outside the abdomen is unduly bulky it may be trimmed under local anæsthesia any time after the fourteenth day. At least ½ in. of bowel

should be left outside the abdomen in relation to each opening. The spur of bowel is divided and then the excess of bowel is removed from the proximal and distal part in turn. For this purpose a pair of straight scissors is the best instrument. Hæmorrhage is usually free and a blanket stitch of catgut is advisable round each orifice; mucous membrane should be included in this stitch. If the colostomy is neat when the time comes for the rod to be removed I think there is no particular object in dividing the spur. The rod is simply slipped out.

(3) *Daily Routine.*

On the day after the colostomy is opened the routine of the daily wash-out is begun. One to two pints of soapy water are run into the proximal opening each morning by means of a rectal tube and funnel. The patient as he lies on his left side has a large kidney dish into which he allows the enema to return. This is done every morning for the rest of his life. The whole performance takes from ½ to 1 hr., according to how soon the wash-out is returned. If the colostomy has been done for an inoperable growth, a wash-through from the distal colostomy opening to the anus is carried out daily or on alternate days according to indications, plain water, hazeline solution, etc., being used.

Patients do extremely well on this routine and often continue for months without any irregular action of the colostomy after the morning wash-out. There is certainly no stasis in their large bowel and I have often been surprised at the extraordinarily clear complexions developed in colostomy patients using this routine.

(4) *Belts.*

The St. Mark's Hospital pattern colostomy belt has no cup. It is not needed. A thin piece of cotton wool about 5 in. square is placed next to the skin, then a flat celluloid plate, 4 in. in diameter, with four studs facing outwards which impinge on the flat colostomy belt.

A flat belt of this kind has many advantages—it is cheaper than most of the more elaborate gadgets; it is easy to keep clean, and it supports the abdominal wall at the site of the colostomy. Indeed, it is not necessarily essential that a surgical belt at all should be worn; some of my female patients have either discarded their belts or have never had one in use. A simple corset such as the "Abdo" costing about 5s., of a stock size, together with the piece of wool and the celluloid plate, is all that is needed. A doctor's wife found this out and I believe it is a very good idea. The corset is quite sufficient for the purpose and its low price places frequent renewal within the reach of those of the most limited means.

(5) *Diet.*

A patient with a colostomy usually has to avoid certain foods which are liable to produce attacks of diarrhœa, such as green vegetables, cabbage, sprouts, etc., currants and raisins, and beverages such as beer and stout. However, one cannot lay down arbitrary rules: the only thing for a patient to do is to experiment and generally avoid things which persistently upset him.

Sir CHARLES GORDON-WATSON, K.B.E., C.M.G.,

said that it was evident after listening to the opening of the debate that excellent results with colostomies could be obtained by different methods as in many other types of operation. For his part he preferred the rectus incision made high, so that

106 Gordon-Watson: *Discussion on Colostomy*

the opening was about the level of the umbilicus and more or less in the middle of the rectus.

He wished to lay stress on certain details. The incision, if made large enough for exploration, must be reduced so that there was only just room for the bowel and glass rod. If the opening was made too large there was considerable risk of a hernia of the small intestine. He had had to deal with a strangulated hernia of this nature as well as chronic hernias. If the opening was too small obstruction might follow,—a danger which was increased by an intermediate stitch. He had been asked to see a case of obstruction which had followed colostomy performed by suturing the skin of the two sides together beneath the loop: the obstruction was at once relieved by dividing the intermediate stitch.

After splitting the rectus fibres he was in the habit of passing a finger up and down over the posterior sheath to displace the intercostal nerves. If a nerve was divided segmental paresis of the rectus might occur and post-operative protrusion was encouraged.

He laid stress on suturing the posterior rectus sheath to the anterior, thus establishing an opening in the split rectus which was bounded by peritoneum, and which encouraged early and strong adhesions with the visceral peritoneum of the loop. At the same time shutting off the compartment of the rectus was a safeguard against the spread of sepsis or hæmorrhage into the abdominal wall should it occur. The deep epigastrics should be carefully avoided or sutured. He left in the glass rod for about three weeks in order to obtain a good spur. He was not in favour of dividing the continuity of the bowel; this he thought promised retraction. One of the causes of retraction of the bowel was contraction of the skin pushing the spur back, and this was sometimes due to too small an opening in the skin at the time of operation, especially when followed by chronic irritation and the formation of a fibrous ring round the colostomy. Colostomies should be kept under observation for some time after operation, so that any tendency to contraction of the skin could be prevented. Mr. Miles's method of removing a circle of skin was, no doubt, designed for this purpose.

Another important detail was the avoidance of tension in the mesentery, which could usually be avoided by mobilization and the division of fibrous bands on the outside of the mesentery which were present in most instances, and which, he thought, were more often developmental than inflammatory, i.e., in the way of the pelvic colon making attempts to obliterate its mesentery in the same manner as the descending colon did. He had twice seen very troublesome hæmorrhage into the mesentery of the pelvic colon following undue tension, and on one occasion a hernia of the small intestine through the hole in the mesentery made by the glass rod and automatically enlarged by tension. It was far preferable to perform transverse colostomy than sigmoid colostomy under tension, and this transverse colostomy was more easily effected through a rectus incision than an iliac.

In his opinion, no sutures should be passed through the bowel-wall itself. These stitches could and did cut out during straining and were sometimes responsible for leakage and sepsis round the colostomy. Experience showed that they were both unnecessary and dangerous.

With regard to the management of a colostomy he was a firm believer in the daily wash-out, which, in the great majority of cases, after a few weeks' experience was rapid and efficient and allowed the patient to follow his daily occupation without inconvenience and often without a thought of his colostomy till the following day. He disapproved of bags and cups, which were unnecessary, disagreeable and harmful. In the great majority of cases a simple dressing with or without a flat celluloid disc and a good belt, such as the St. Mark's Hospital pattern, was all that was required to

keep the patient both comfortable and clean. Cups caused congestion and prolapse, and were uncomfortable and often offensive.

He thought that it was time that emphasis should be laid on the fact that colostomy was not the dreadful operation it was supposed to be by the profession and the public. If a carcinoma was inoperable colostomy should always be recommended both to prolong life and to make the patient more comfortable, though amongst the poorer classes difficulties arose in the management of colostomies.

A colostomy properly performed and properly managed was one of the most satisfactory palliative operations in surgery. Patients could carry out their duties in any walk of life. He instanced the case of an engine-driver on the Great Western Railway, who had continued at his post with a colostomy since 1911.

Mr. R. P. Rowlands, O.B.E.

(1) Indications.

I believe that when well performed for carefully selected cases colostomy is a valuable operation, and that the public and professional fear and dislike of it are not justified. It prevents suffering, and saves or prolongs many lives, especially when used either as a temporary or permanent measure in certain cases of cancerous obstruction of the pelvic colon or rectum.

It is particularly valuable in relieving the obstruction, spurious diarrhœa, or incontinence, of irremovable cancer of the rectum, and in correcting intestinal obstruction, cystitis, fever, and ascending nephritis associated with colico-vesical fistula caused by diverticulitis of the pelvic colon.

It is more effective than cæcostomy in relieving distension of the colon due to complete obstruction low down in the colon or rectum. Therefore it is a better preparation to resection of an obstruction in this situation. In such cases colostomy is invaluable as a temporary and life-saving operation.

But permanent colostomy is undesirable whenever either resection or short-circuiting can be carried out without undue risk. For instance an irremovable growth of the splenic flexure or descending colon is not well treated by permanent colostomy, which can be avoided by a short-circuit between the transverse and pelvic colons.

(2) Technique.

I consider a small parietal opening essential, and above all I never bring out the colon through the exploratory incision so that the following complications may be avoided: (*a*) ventral hernia; (*b*) prolapse of the small intestine; (*c*) infection of the wound, and (*d*) complete lack of control of the artificial anus.

High left inguinal or iliac colostomy is the most satisfactory in my experience, and should be adopted whenever possible. I prefer to bring the loop of the colon outside the body and to hold it up with a glass rod, with rubber tube attached. Failing the glass rod, small rubber-covered artery forceps are good substitutes, which are always available. A single suture at each angle of the skin incision is often necessary and the ends of each suture can be tied to an appendix epiploica in order to fix the colon. It is important to make the opening in the highest available part of the left colon so that prolapse of the mucous membrane may be avoided.

When it is urgently necessary to open the bowel during the operation, I insert an air-tight manner a rubber tube, which is more comfortable and remains in for a

longer time without leakage than does a glass tube. If there is difficulty in delivering the selected loop of the colon, division of the left parietal peritoneal reflexion generally overcomes such difficulty, and this plan is much better than suturing the imperfectly delivered colon to the parietal peritoneum—a method which has led to leakage of fæces into the peritoneum, because the stitches have been torn off during vomiting or coughing.

Although an end-colostomy is very satisfactory there is rarely any advantage in dividing the colon and closing one end; the wrong end has unfortunately been closed in some cases. A good spur can be obtained by the glass-rod method, especially if the rod is not removed too soon but is left in for at least ten days.

As regards obtaining a controllable artificial anus, I believe the most important point consists in the use of a small or stab parietal wound. Efforts to make a valve often defeat themselves and lead to unsatisfactory emptying of the colon with too frequent and too small actions.

Mr. Lionel E. C. Norbury, O.B.E.

My own practice in cases of carcinoma of the rectum is usually to perform a a sub-umbilical colostomy through the fibres of the left rectus muscle. In my experience the patient obtains a better control over the artificial anus by this method than is the case with an inguinal colostomy. He is also able to control the passage of flatus to a certain extent by putting the rectus muscle into action when he feels that wind is passing.

I employ a glass rod passed through the mesocolon in order to get an efficient spur, and the rod is not removed for at least fourteen days. I usually suture the sero-muscular coat of the bowel to the opening in the peritoneum, and do not as a rule divide the bowel completely across when the glass rod is removed.

Complications.

I have met with several, regarding the treatment of which I should be glad to have the opinion and advice of my colleagues.

(1) *Retrograde intussusception of the lower end of the colostomy with gangrene of this portion of the bowel.*—This occurred in the case of a lady several months after the performance of colostomy for inoperable carcinoma of the rectum, necessitating amputation of the intussuscepted portion.

(2) *Contraction of the colostomy opening with the formation of a tight fibrous ring producing obstruction and necessitating the fashioning of a new colostomy.*—This complication arose in a patient whose rectum I had removed by the perineal route some twelve months previously. Even after the formation of a new and apparently satisfactory artificial anus, there is still a tendency to contraction, and now some three years after the original operation, occasional dilatation of the stoma is necessary.

(3) *Rupture of a diverticulum of the pelvic colon in a case of acute intestinal obstruction due to carcinoma of the rectum.*—This occurred in the case of an elderly gentleman as I was bringing the loop of pelvic colon to the surface. Several diverticula were present and one of these gave way during manipulations. I therefore utilized this opening in the bowel in order to tie in a Paul's tube. It is important to bear in mind this possible complication in such cases of acute intestinal obstruction.

(4) *Prolapse of the bowel at the colostomy opening.*—This is difficult to prevent, for if one utilizes the upper portion of the loop for the colostomy the loose lower

portion may prolapse, and vice versa. Again, if the pelvic colon has a long mesentery and a colostomy is effected at the middle of the loop, then both the upper and lower ends may prolapse. Such a complication is most distressing to the patient. Allingham, in his work on "Diseases of the Rectum," recommended that in cases in which a long pelvic mesocolon is present, the whole loop of bowel should be withdrawn from the abdomen, opened some twelve to twenty-four hours later, and the redundant loop excised when the bowel is firmly adherent to the abdominal wall.

Mr. F. Swinford Edwards

said that the aim of the surgeon when performing colostomy should be to prevent the passage of bowel contents from the proximal to the distal portion of the sigmoid, and, at the same time, to prevent any prolapse of small intestine through the wound—an accident generally attended by a fatal result. Both these aims were best obtained by the formation of an effective spur through inserting a deep-buried silkworm-gut suture which bisected the wound. A glass rod might also be employed—where the operator wished to accentuate the spur—but the deep suture should never be omitted.

The surgeon's third aim was to confer as much control as possible, and this was best achieved by hypergastric colostomy through the left rectus muscle. He (the speaker) had introduced this method at St. Mark's Hospital about twenty years ago, and he was glad to hear that it still continued to be the one most generally practised there. He (Mr. Edwards) was opposed to the employment of all cups, plates, bottles, and bags in the after-treatment as being absolutely unnecessary and even harmful, after a properly performed colostomy.

Mr. J. P. Lockhart-Mummery (President)

said that as regards the technique of the operation he believed that the rectus incision high up in the abdomen was far superior to any other. He was convinced that it gave at least 50 per cent. better control over the opening than the old-fashioned opening in the left iliac fossa. His experience was that a modern colostomy opening done through the rectus muscle supplied an amount of control over the evacuations, both of fæces and flatus, which was sufficient in the great majority of persons to enable them to live quite ordinary lives in comfort and security from possible unpleasant accidents.

He had not found that a daily wash-out was necessary in most cases. His own technique was of the simplest. He used a small incision and sutured only the anterior rectus sheath and the skin, and relied on a glass rod to form the spur; he was completely satisfied with the results. The chief difficulty that might be met with was when the patient was abnormally fat. In such cases he believed the best practice was not to attempt to bring the gut to the surface, but to take the surface down to the gut. He cut away a large area of fat and allowed the skin to come down to the aponeurosis. By this method he had successfully got over difficulties arising in the case of patients with very fat abdominal walls. As regarded suitable apparatus he believed that a simple belt and a flat celluloid disk gave by far the best results.

[A number of drawings were shown illustrating the development of colostomy from the time of Amussat's operation down to the modern colostomy through the rectus muscles.]

110 Milligan—Fitzwilliams: *Discussion on Colostomy*

Mr. E. T. C. Milligan.

Although it is my practice to perform colostomy through the left rectus muscle because of its advantages, I am, nevertheless, conscious of certain drawbacks to the method. (1) Bleeding from the deep epigastric artery and vein often causes some embarrassment in a small incision, and secondary hæmorrhage from these vessels has occurred. (2) Ventral hernia is seen from time to time. (3) The umbilicus, being situated so close to the colostomy, requires special attention for cleanliness.

I do not believe that a patient can voluntarily control his colostomy by muscular action wherever it is situated. Indeed, there can be no warning sensation from the insensitive colon to induce sphincteric action, nor is the normal tone of the abdominal muscles round the colostomy an adequate sphincter. Nevertheless, it should be more widely recognized that a colostomy can be satisfactorily controlled by two simple means, without the employment of which the life of a colostomy patient may be a burden to him: (*a*) A morning colon wash-out. (*b*) Prevention of soft or fluid fæcal residue in the colon, attained by the avoidance of excessive ballast-containing foods and aperient medicines.

That colostomy is unsuited for persons in all walks of life in spite of the eulogistic accounts given by previous speakers, was impressed upon me by the fate of two patients with well regulated colostomies, who soon after leaving the convalescent home, terminated their existence by violence. They discovered that their colostomies handicapped them in the search for employment, and that the extra attention their daily evacuation called for, was ill suited to a roaming existence in lodging houses.

Therefore at any rate in the case of hospital patients the circumstances should be investigated, and if colostomy is inevitable, provision should be made for the patient's future.

Colostomy, excellent measure as it is, is still a very high price to pay for the cure of carcinoma of the rectum, and it would be a pity if we, as specialists in this subject, rested content with the present position of operative treatment, simply because the recurrence rate is not high, and because some persons of leisure and in comfortable circumstances, or taking a fatalistic view of life, declare themselves satisfied with a colostomy. We must aim at the cure of the condition without the sacrifice of so important an organ to our well-being in life as the rectum.

Nor do I believe that colostomy should be performed, as some say, in all cases at the first possible moment that the malignant growth in the rectum is declared inoperable. I have known cases of inoperable carcinoma of the rectum in which the only inconvenience was slight bleeding at stool. On the other hand, cases occur frequently enough in which carcinoma has existed for years without much inconvenience to the patient.

Surely life without a colostomy is better than a slightly prolonged existence with one.

I nevertheless advise a patient with inoperable carcinoma of the rectum to undergo colostomy when his present symptoms are such that they would be benefited.

Mr. Duncan C. L. Fitzwilliams, C.M.G.,

said that it was quite obvious from what the previous speakers had said that there was more than one way of performing colostomy in a satisfactory manner. He had carried out the operation both through the rectus and in the lateral part of the abdominal wall, and he did not find there was much to choose between them; if anything,—on theoretical grounds, better muscular control was to be obtained through

a grid-iron incision high up in the lateral part of the abdominal wall and that was the method he had adopted for some time. A pad, too, worn over the colostomy opening appeared to him to sit better if it was steadied by the anterior superior spine of the ilium, and less liable to slip than when placed over the more anterior and mobile parts of the abdominal wall.

He thought that surgeons, and even hospitals, sometimes followed fashions in operating. It was quite obvious that the staff of St. Mark's Hospital all performed the operation through the rectus. The habit of the surgeon rendered the operation easier and more familiar to him and other methods might then be thought inferior.

Mr. Several surgeons had spoken of the desirability of making as small an opening as possible, but there were a large number of cases of rectal cancer in which the operation seemed feasible from a rectal examination. But he supposed that no surgeon would perform such an operation without previously having examined the liver, for if there was a secondary growth in the liver, the patient's life would not be prolonged a single day by excising the rectum, and he might just as well be saved such a serious and unpleasant operation. The discovery of a secondary nodule absolutely negatived any such operation. How could such an examination be made through a small incision? It was quite impossible. Mr. Miles was the only surgeon who advocated a separate incision for examination purposes, therefore a small incision was in most cases inadequate. He never made an attempt to perform the operation by a small incision, but always used one large enough to admit the whole hand so as to examine both lobes of the liver. The bowel was then brought up, mobilized if necessary, and a glass tube inserted; a silkworm-gut suture was always put in through the muscles, the mesentery, and through the opposite muscles and back again, and tied tightly. This made the spur permanent and also prevented prolapse and subsequent hernia, in fact he had never seen a hernia in any of his cases.

In passing the suture it was advisable to push the blunt or eye end of the needle through the mesentery rather than the sharp point, as with the point vessels might be pierced; before he himself had adopted this manœuvre he had seen large and troublesome hæmatomata develop.

The muscles were always stitched tightly round the bowel and the appendices epiploicæ incorporated in the knot. This procedure fixed the bowel quite sufficiently.

He had never cut out any skin, nor did he think it necessary when a large incision was used, but in small incisions it was probably advisable. In any case, stricture of the opening, later on, was always due to contraction of the skin in a small opening, and was relieved by excising skin.

He usually cut off the bowel externally, very freely, and in those cases in which further operation was considered inadvisable, he pulled out as large a loop as possible and removed it flush with the surface. In one case he remembered that the bowel became gangrenous either from the muscles being sewn up too tightly, or from the stitch through the mesentery interfering with the circulation. It was cut off as soon as this had become apparent and an excellent result followed.

The operation was also very often performed for the relief of acute obstruction, and in these cases a Paul's tube was necessary. He always used a modification of Paul's tube which he had brought out some years before the war. It was fitted with a side-way to which a small rubber tube was attached which projected above the dressings. This enabled the bowel to be flushed out without disturbing the dressings, when the large exit tube was occluded. It also enabled fluid to be administered and to be absorbed by the patient, thus replacing that lost by prolonged vomiting. It also had another effect, for which indeed it had been invented, namely, to introduce glucose into the bowel as a food for the patient. Many of the patients

were old and feeble, and had been unable to keep anything in the stomach for some days, and if the anæsthetic upset them, actual starvation became a factor which in these cases was not sufficiently realized, and was the cause of death in some of more feeble subjects. The glucose in those days had to be obtained direct from the brewers and was unknown in hospitals.

He was very interested to hear of the patient who evacuated his bowels once a week, as he remembered Professor Caird telling his class of a woman who trained her colostomy opening to inactivity for six days, and used to retire on the seventh, and spend her time clearing herself out completely. She carried out her household duties and looked after a large family.

Mr. Miles (in reply)

repeated his axiom that it did not matter what method was used provided the colostomy was effective. It was a great mistake to delay it in cases of carcinoma, every day of delay was a day to the bad. His objections to the rectus method were that it required considerable length of colon and produced a transverse fold within the radial range of the small intestine.

Section of Surgery.

President—Mr. V. Warren Low, C.B., F.R.C.S.

DISCUSSION ON THE SURGICAL TREATMENT OF CHRONIC NON-TUBERCULOUS ARTHRITIS.

Dr. A. P. Beddard

said that it was generally admitted that rheumatoid arthritis and polyarticular osteo-arthritis were low-grade septicæmias, comparable with gonorrhœal rheumatism—that was to say, the joint changes were primarily infective—but he doubted whether the same was true of monarticular osteo-arthritis, in which, although there was generally an infective element, there were other factors present as well. As for bacteriology, he believed the micro-organism concerned to be *Streptococcus longus*, more often of the *viridans* than of the hæmolytic type.

Years ago he saw cases which were undoubtedly pneumococcic in origin, but for some years past in England, until recently, the pneumococcus had been more conspicuous by its absence. He looked upon rheumatoid arthritis as certainly septicæmic. The primary sites of infection were the teeth, tonsils, sinuses, alimentary canal, including gall-bladder, the male urethra, and the uterus.

He had occasionally seen fairly acute cases in which no site of infection could be found, and it might be noted how symptomless chronic infection of teeth, tonsils, and sinuses might be, and how small a local trouble could keep up joint changes once the process had been started.

There were two sites of infection, the treatment of which was very unsatisfactory. One was the uterus, and for a chronically infected endometrium there were but two treatments; curetting, which in his experience was perfectly useless, and removal of the organ, which was a confession of failure. The other unsatisfactory site was the alimentary canal, and here neither vaccines, nor drugs, nor diet, nor the Plombière's treatment could be guaranteed to put an end to the infection. Surgery was very occasionally of value for the purpose, but it was not infrequently applied without discrimination. On rare occasions he had seen a chronically infected gall-bladder which had resisted medical treatment, removed with advantage. He imagined that it was true of any tube in the body that if it was infected and there was delay in the passage of material along it, there never would be an end to the infection so long as that delay existed.

Speaking generally, he thought the medical treatment of early cases of rheumatoid arthritis and polyarticular osteo-arthritis was fairly satisfactory, and that the progress of the disease could often be arrested and the patient be left with a fairly useful, if slightly deformed, joint. The signs of failure were persistent pain and deformity. Speaking as a physician, he had certain cases in mind which were medical failures, namely, cases of rheumatoid arthritis in which infection had not been controlled, the joints being liable to periodic acute outbreaks of inflammation, and cases of rheumatoid arthritis or polyarticular osteo-arthritis in which infection had been controlled, and the joint was partially movable, but in which any except the most limited movement gave much pain.

114 Page: *Surgical Treatment of Chronic Non-Tuberculous Arthritis*

Mr. C. Max Page, D.S.O.

It is my duty to define, as far as possible, the limitations imposed by the title. Despite the omission of tuberculous conditions, and by implication, pyogenic infections, a wide and ill-defined field is left open for discussion.

It would, of course, be satisfactory to base one's classification of chronic arthritis on its pathology, but I do not consider this is a practical proposition. The ætiology of arthritis not due to recognized specific organisms remains obscure and confused. No doubt there are reasonable theories to account for the various conditions observed, and very elaborate classifications have been drawn up, based in the main on the anatomical findings. Accepting the limitations imposed by our basic ignorance of the ætiology, I propose to consider chronic arthritis for the purposes of this discussion under the two commonly accepted groups, namely: (1) Rheumatoid arthritis; (2) osteo-arthritis.

(1) *Rheumatoid Arthritis.*—It must be admitted that several different infections are grouped under this heading, but the clinical manifestations as a whole suggest that rheumatoid arthritis is due to a bacterial invasion, almost comparable in its active stages to a chronic pyæmia. During the period of activity the surgeon has little scope for action, except so far as he directs measures calculated to prevent the development of deformity. Occasionally a single joint is affected by this form of disease, and then early direct surgical interference may be justifiable and useful.

(2) *Osteo-arthritis.*—In regard to this second group, it would appear to be generally agreed that the joint changes are produced as a reaction to circulating toxins, rather than to the presence of micro-organisms in the joint membrane. I do not propose to discuss the sources of these toxins, though I would make it clear here that the surgical treatment of any example of osteo-arthritis should be preceded as a matter of course by the elimination, as far as is possible, of the source of these toxins by medical or surgical means. Again, trauma in its widest sense is undoubtedly a factor in most cases. In practice it does not appear to me that the actual anatomical form an osteo-arthritis assumes has a very important influence on treatment, and I will defer any reference to the sub-classifications of the condition till I deal with the individual joints.

Dr. Beddard has indicated the preliminary medical treatment in both groups of cases, and has shown where the surgeon may properly take part in the treatment of the cases. I shall therefore proceed to define first, the conditions which, in my opinion, justify operative treatment, and then consider in outline the technique and methods adapted to particular joints under various conditions.

As stated above, the surgeon is seldom asked to deal with subacute polyarticular infections. Occasionally, on account of pain or progressive deformity in one joint, action may be called for. The form it should take may simply consist in fixation by splintage, possibly preceded by manipulation. Sometimes an excision of the joint surface or of the synovial membrane of one or more joints may appear worth carrying out. It is, however, mainly in the field of osteo-arthritis that the surgeon has his opportunity. I would put down the indications for operative interference in the latter condition as follows:—(*a*) For derangements of joint action secondary to the disease process; (*b*) for joint pain; (*c*) for progressive deformity.

In the first group manipulative treatment may be tried sometimes with success, especially when painful limitation of movement is in evidence. I shall leave it to Mr. Elmslie to define the scope and details of this method of treatment. When symptoms of internal derangement are present, the operation will, as a rule, be in the nature of an exploratory arthrotomy. The joint is fully explored by a suitable

incision, detached osteophytes or proliferated synovial membrane which interfere with joint movement are removed, and the articular surfaces are perhaps trimmed. Generally, I have been surprised at the degree of improvement which often follows operations undertaken for this condition, both in regard to function and in the loss of pain. Operations of this kind cannot, however, be said to be radical, and in cases in which the disease is advanced, prove of only temporary benefit. If pain and deformity are prominent features, more extensive procedures must be carried out. Such operations will fall into one of the following types: (*a*) Synovectomy; (*b*) arthrodesis; (*c*) arthroplasty; (*d*) simple excision of the joint.

The choice of operation is less dependent on the anatomical condition of the joint and on the assumed ætiology than on the age and general condition of the patient. With the young and middle-aged the aim should be not only to remove pain, but to provide a stable joint, at any rate in the case of the lower extremity. This can of course be most certainly effected by establishing an arthrodesis, and, in the case of the knee and hip, I have a strong leaning towards this procedure. The more conservative operations, which aim at the production of a fibrous joint or a new joint, are naturally more attractive to the patient at the time, but clearly leave him open to a recurrence of the original process. In the elderly and enfeebled the procedure to be preferred is one that will guarantee relief from pain while entailing a short operation and simple after-treatment.

In order to estimate the relative merits of different operative procedures the after-history of considerable groups of cases is necessary. Evidence of this nature is still very scanty. Such as it is I shall refer to it when discussing the treatment of individual joints.

I will now discuss the individual joints, and mention points in relation to each which may justify radical operation, and indicate the operative methods most suited to each.

In selecting the ideal operation for the joints of the upper or lower extremity, the conditions under which they have to work must be clearly appreciated. If a joint is not submitted to any continuous strain, free mobility may be allowed without the probability of progressive absorption of the joint surface, although its surface be denuded of cartilage. This condition rules in the joints of the upper extremity. On the other hand, the large joints of the lower extremity are necessarily exposed to continuous pressure, and for this reason any reconstruction of the joints must be planned to produce a condition able to withstand continuous weight-bearing.

The joint upon which our practical knowledge of operation results is chiefly based is the metatarsal phalangeal joint of the big toe. It has been found by experience that the free excision of the head of the metatarsal gives, as a rule, a mobile and painless joint. Observations over a period of years have been made in these cases, and I think this conclusion is generally accepted. How far these results can be applied to other joints is perhaps doubtful, because the new joint produced in this way is exposed to very little pressure or strain at the joint surface. The deductions to be drawn from the results of operation on this joint are further complicated by the fact that they are carried out as often for deformity as for an arthritis.

The Hip-joint.

The pathological condition of the joint associated with severe symptoms varies widely. The X-rays apparently always show reduction in the normal inter-articular space on account of the absorption of the joint cartilage. The amount of bony deformity may be slight or gross. In some cases the bony change is hardly recognizable, and the symptoms are often due to a chronic inflammation, very

116 Page : *Surgical Treatment of Chronic Non-Tuberculous Arthritis*

possibly of bacterial origin. It should be noted that the same case, if left untreated, will gradually develop appearances indistinguishable from chronic osteo-arthritis.

The operations which have been commonly performed for the condition are :—

(1) Arthrodesis.
(2) Arthroplasty.
(3) Partial excision of the joint.

(1) *Arthrodesis.*—Albee introduced arthrodesis in 1918, and has since carried it out in operations on a large number of patients. He reports favourable results

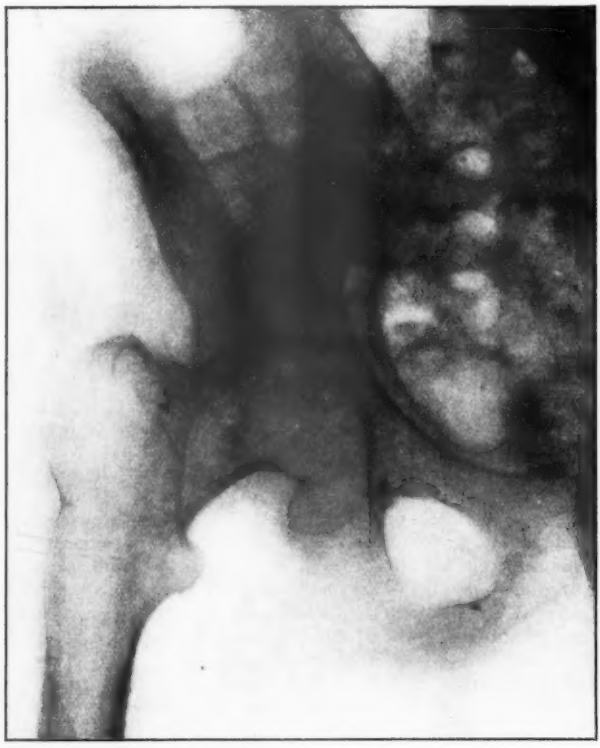


FIG. 1.—Firm bony ankylosis of hip-joint following an arthrodesing operation in which the head of the bone had been dislocated. X-ray taken one year after operation.

in most cases. I have carried out the procedure in some seventy cases, and can confirm this view. It should, however, be said that bony ankylosis is not always obtained as a result of the operation as usually carried out, a fibrous union permitting of some movement being a fairly common result (figs. 1, 2 and 3). In all cases the original articular pain is relieved.

(2) *Arthroplasty.*—My experience of this procedure in this joint is limited to five cases, and the results have not been very promising. In no instance was a full range

of movement obtained, and in most cases the patients complained of some pain afterwards. The operation I carried out was that of dislocating the joint, erasing the surface of the head of the femur and capping it with a covering of fascia lata. The capped head of the femur was then returned to the acetabulum.

(3) *Partial Excision of the Head of the Femur.*—This procedure can be rapidly carried out and leaves a movable joint. The degree of mobility varies in accordance with the amount of the head and neck removed. If the stump of the neck is returned to the acetabulum, and the thigh maintained in the abducted position for a few weeks

FIG. 2.—Bony ankylosis of hip-joint associated with some new bone formation above old joint-line. X-ray taken three years after operation.

after operation, relative stability is assured. Platt has carried out this operation frequently, and is impressed with the favourable results obtained in the early period, but has noticed the liability to a recurrence of pain later in the history of the case. It is an operation of which I have only a small experience of eight cases. It would appear to be a sound measure to adopt for elderly people or in instances in which both joints are affected by the disease process. It is simpler in execution than an arthroplasty, and equally effective in the relief of pain and provision of movement. A criticism which may be levelled against the method is that the shortened neck of

the femur leads to limitation of abduction and a tendency to an adduction deformity. The first is due to impaction of the great trochanter on the margin of the acetabulum, and the second to the reduction of the lever through which the gluteal muscles act. These shortcomings can be overcome by adopting the procedure advocated by Whitman, namely, that of detaching the great trochanter, and re-applying it lower down on the outer surface of the shaft of the femur. An operation based on these

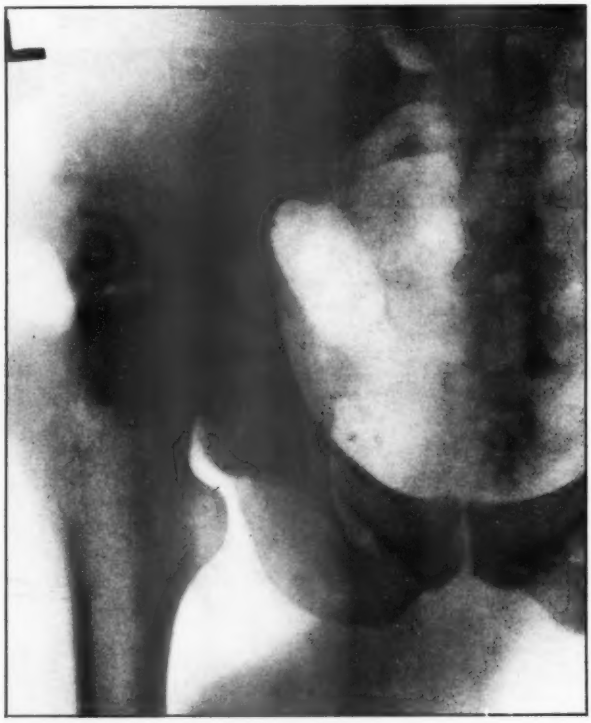


FIG. 3.—"Fibrous joint" following complete operation for arthrodesis. X-ray taken four years after operation.

lines is being tried by Bristow and others, and is given the name of reconstruction of the hip-joint.

Bilateral cases present an awkward problem. I have usually carried out an excision or arthroplasty on one side, and then fixed the other. It would perhaps be best to excise on both sides.

Technique of Operation.

A satisfactory exposure of the hip has been the main difficulty in these operations. After the trial of several routes I have come to the conclusion that an external approach based on Murphy's method is the most satisfactory (figs. 4 and 5). The

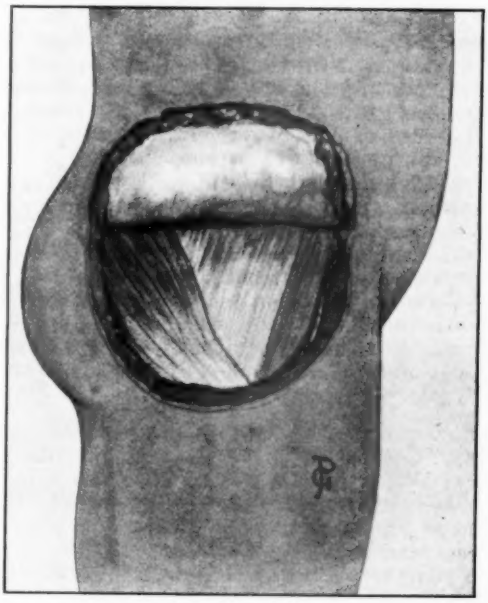


FIG. 4.—The skin incision employed in approach to hip-joint in arthrodesing operation.

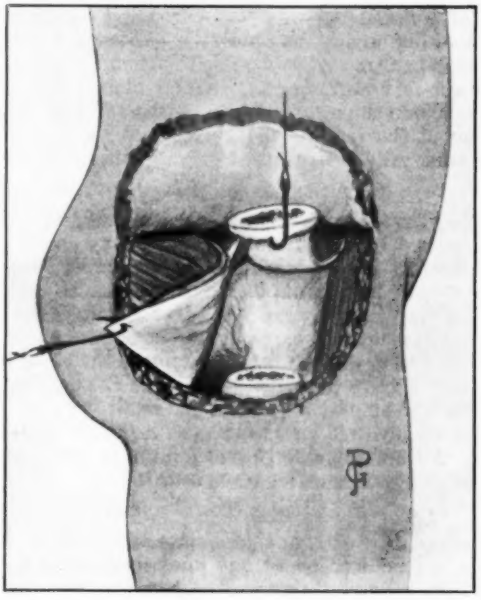


FIG 5.—Diagram of the exposure of the hip-joint given by this approach.

120 Page: *Surgical Treatment of Chronic Non-Tuberculous Arthritis*

method is a rapid one and is associated with very little bleeding. In most cases, then, exposure allows of dislocation of the head of the femur backwards, after incision of the capsule. This approach permits any of the operations described above to be carried out in a short time. After the operation the great trochanter is usually sewn back in position and in cases of excision of the head it can be planted lower down on the shaft of the femur as suggested by Whitman.

After-treatment.—If an arthrodesis is aimed at, the patient is put up in a plaster spica for six or eight weeks. After this a short hip spica is worn for another month, and the patient allowed to walk on crutches and put the weight through the leg operated upon. In the case of elderly patients, or of those upon whom only a partial excision or an arthroplasty has been attempted, the limb is slung after operation in a Hodgen splint. At the end of five or six weeks, or even sooner, they can get about with crutches, and commence to put weight through the operated limb two or three months after operation. The end-result cannot be expected under six months and the function of the limb often improves up to one or two years after operation.

The Knee-joint.

This joint is more commonly the site of noticeable osteo-arthritic change than any other in the body. Commonly, the symptoms are chronic synovial effusion or mechanical disability. When chronic thickening of the synovia is a prominent feature, and when the articular surface shows little change, as may be the case in the condition known as lipoma aborescens, the operation of synovectomy may be carried out. The procedure requires an incision similar to that employed for an arthroplasty of the joint. In this way it is possible to remove the bulk of the synovial membrane. The late results seem fairly satisfactory as far as the range of movement is concerned. I have only observed three cases for four years or more. In these there has been no progression of the disease, though symptoms similar to those of a mild osteo-arthritis remain.

For symptoms of internal derangement suggesting catching or locking of an osteophyte, an exploratory arthrotomy may be carried out. The results of these operations are, in my experience, more satisfactory than one would expect. I am not clear what the prognosis is, however, and I shall be interested to hear other speakers' views on this point. When the disease is advanced I think there can be no question that a simple excision of the joint is the only sound method of effecting a radical cure. At any rate, personally, I have not tried any other method.

Operation on the Knee-joint.

In excision of this joint for osteo-arthritis, I have adopted a method which has given satisfactory results. By the ordinary method of excision and fixation sound bony union is not always obtained, a fibrous joint being formed which may be painful. Certainty of bony union can only be secured if the bony surfaces are kept in close apposition for operation. By rawing the cut surface of the patella and pinning it down to the tibia, fixation of this character is obtained. The limb is easier to handle after operation, and in no case in which this method has been employed has union failed to occur. I have used bone pegs to fix the patella, but found that the most secure procedure is to employ a French nail (fig. 6).

When both knees are involved in the process a double excision seems rather a severe undertaking. An arthroplasty of one joint may yield better results, but I have no experience of this procedure for osteo-arthritis.

Ankle- and Tarsal-joints.

Serious painful chronic arthritis of these joints is difficult to deal with by operative measures. The radical fixation of one joint necessarily imposes strain on the

neighbouring joints not so treated. An arthrodesis of the ankle-joint is sometimes satisfactory, but more often than not pain is developed in relation to the tarsus a few months after walking has been resumed. I think that possibly one may have acquired an unjustifiably gloomy view and outlook with regard to these cases from the experience of post-war injuries. I hope the matter will be referred to by those who follow me in relation to this point.

Sacro-iliac Joint and Spine.

Osteo-arthritis in these areas is relatively uncommon, and in my small experience has not been very tractable to surgical procedures. I shall leave the subject to those who follow me.

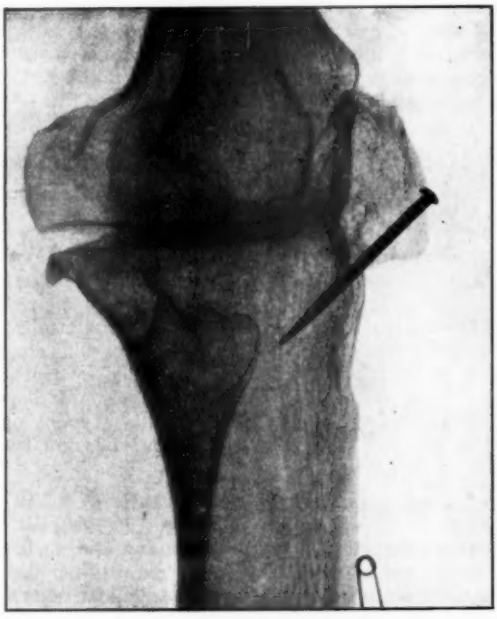


FIG. 6.—Radiogram of a knee excised and fixed by patella-pegging. X-ray taken six months after operation.

Upper Extremity.

It is seldom that osteo-arthritis of the shoulder-joint justifies a radical operation. Many cases of this kind can be benefited by manipulative treatment. If an operation is decided upon, I think a conservative excision of the head of the humerus promises the best result.

Elbow-joint.

This joint is often enough the seat of arthritis associated with an old injury. I have attempted an arthroplasty in cases of this character and on the whole the results are fairly good so far as the relief from pain is concerned. The stability of the joint is variable, but if the olecranon and coronoid processes are retained, a very fair new joint should be produced.

122 Page—Elmslie: *Treatment of Chronic Non-Tuberculous Arthritis*

Carpus.

Chronic arthritis in these cases may be divided into two types. In the first—rarefied osteitis of the proximal row of bones—it may be mistaken for tuberculous development more or less spontaneously. In the other group the arthritis is definitely post-traumatic and follows such injuries as ununited fracture of the scaphoid or luxation of the semilunar. Operative treatment for both conditions consists in excision on the proximal row of the carpus. This allows a new joint to be formed between the head of the os magnum and the lower surface of the radius. Should the os magnum be involved in the disease process a more extensive resection will be necessary.

In my experience the results of these operations have been somewhat uncertain. If much strain is imposed on the joint later, a recurrence of symptoms of the arthritis is common, and in one or two cases I have carried out a complete excision of the carpus at a second or third operation on account of pain. Pain experienced in this joint is at times usually severe and disabling, and the patient is found to be willing to undergo any treatment which may promise relief.

Metacarpal Joints.

Of the small joints of the hand which are subject to osteo-arthritis, that which best repays operative treatment is the metacarpo-carpal joint of the thumb. The condition may be originated by an injury, such as a Bennett's fracture, or arise apparently spontaneously. A subperiosteal section of the base of the metacarpal has given good results in four cases, from the point of view of relief of pain. The function is not materially interfered with if the insertion of the tendons is left intact.

In conclusion, I must apologize for the sketchy nature of my review. The subject, when one comes to face it, is a large one, and full of minor pitfalls. These difficulties arise partly from our ignorance of the pathology of the various conditions, and partly from the uncertainty of the results which are obtained by conservative treatment. Surgical treatment of these joint conditions is a relatively new development, and we still lack decisive information as to the ultimate results of many operations. I hope this discussion will, at any rate, help to crystallize our ideas on the possibilities and limitations of surgery in this field.

Mr. R. C. Elmslie, O.B.E.

Chronic non-tuberculous arthritis includes so many and such diverse conditions that it is evident that in a short discussion only general principles can be dealt with. There are such wide differences in pathology, in mechanical results, and in prognosis between a polyarticular rheumatoid arthritis and a monarticular osteo-arthritis that it is even difficult to lay down general principles of treatment suitable to the two conditions. The subject is rendered more complex by the fact that a monarticular arthritis may be due to one of many known infections, e.g., the pneumococcus or the gonococcus ; to a chemical irritant as in gout, or to a mechanical cause as in a damaged or deformed joint.

The surgical treatment of an arthritis may aim at the cure of the active disease, or at the relief of pain, or its object may be the improvement or restoration of functional use. Incidental to these last two objects the immediate aim of treatment may be the prevention or correction of deformity, or the improvement in or the complete abolition of mobility.

The cure of most of the forms of chronic arthritis remains for the most part in the department of the physician, but the surgical principle of rest for inflamed structures requires emphasis. The use of extensions, splints, and plaster of Paris cases during the more active period of the disease is valuable, not only as a means of preventing

deformity, but also as a means of diminishing the activity of the inflammation. The fear that rest and immobilization of a joint will give rise to stiffness is widespread. It is not, however, the immobilization that produces the stiffness, but the inflammatory changes themselves, and if immobilization will cut short the inflammatory period, the resulting stiffness will be less rather than greater. The principles guiding the best position for resting the individual joints have been very clearly laid down by Sir Robert Jones, but there is one point in which, I think, splinting often requires more care, and that is, in ensuring that no joint should be fixed in an extreme position. The knee should be five degrees short of full extension; the wrist should be dorsiflexed without being forced back; the fingers and thumb should be rested in the position that they assume when grasping an orange or large egg, and not in full extension. An extreme position is uncomfortable to the patient and delays the subsequent restoration of movement.

Aspiration of a joint as a mode of treatment is perhaps neglected. Excessive tension within a joint may be dangerous; in certain joints a pathological dislocation may occur. I have seen three instances of dislocation resulting from pneumococcal arthritis of the hip within a year, and it would seem that such results could be prevented if the condition were recognized and the joint either aspirated or incised.

In the prevention of deformity by splinting, great difficulty is found in some forms of arthritis from the presence of severe muscular spasm, which not only renders the use of an extension, or of a splint, intolerable to the patient, but also makes it very difficult to eliminate the occurrence of pressure sores, because it is impossible to avoid getting uneven pressure from the splint. In such cases a plaster-of-Paris case extending widely above and below the joint, applied whilst the patient is anæsthetized, is in my experience often the only safe way of preventing contracture. It is sometimes justifiable to perform a tenotomy or a partial neurectomy in order to diminish such spasm (e.g., of the adductors of the hip or of the hamstrings).

The relief of pain is often the most important object in the treatment of arthritis. All the methods already mentioned may help towards this end, but there are many monarticular cases in which pain persists for years with a slowly progressing deformity and loss of mobility; this, in the end, leads to more drastic surgical procedure. A fixation splint on the knee, or a weight-relieving caliper on the hip helps for a time, but the condition progresses and the appliance is irksome. In such cases fixation of the joint by operation (arthrodesis) is called for. The efficacy of this operation in the case of the hip is now very generally recognized, but we still see patients too often with a painful, stiff and adducted hip, or a flexed and stiff knee, in which all movement has been lost, going from one form of medical or physical treatment to another in the hope of a cure, when an arthrodesing operation would render them comfortable in three months.

The surgical methods that may be used in attemping to improve function are many and diverse. First among them may be put manipulation of the affected joint or joints whilst the patient is anæsthetized. A clear distinction must be drawn between a manipulation performed with a view to correcting a deformity, which may be carried out whilst the arthritis is active and which is followed by fixation in the new position, and a manipulation, the object of which is to try to increase the range of movement of a joint. This latter manipulation can only be safely undertaken during a quiet period, and great care and experience are required in selecting cases that will benefit. The monarticular arthritis without much pain or bone change as seen in the X-ray, but with certain movements restricted and painful if forced, is the most favourable type; in such, as a rule, the movement may be forced with considerable freedom. In monarticular arthritis with greater restriction of movement manipulation will often help, but care must be taken to see that the active

inflammatory stage is over; the manipulation should not be too forceful, it being only necessary to attain a moderate increase in the range of movement. Used carefully and at the right time such manipulation may even help to restore mobility in the worse type of gonococcal arthritis and in puerperal arthritis (provided that X-ray examination does not reveal too great destruction of the joint surfaces).

In polyarticular arthritis of the rheumatoid type manipulation may sometimes help to improve mobility. It is my practice to carry out this only at the immediate suggestion of the physician who has been watching the case, and who is therefore able to judge of the activity of the inflammatory process.

A second item in the surgery of the restoration of function is the removal from the joint of such mechanical obstructions as loose bodies, or, in the case of the knee, torn semilunar cartilages. Such removal, besides helping to restore the more normal use of the joint, also assists in curing the arthritis. A knee-joint in which there is a torn cartilage may show extensive osteo-arthritic changes, which clear up to a considerable extent when the cartilage has been removed.

The practice of removal of osteophytes, and trimming down of the margins of the articular surfaces, is useful in certain selected cases. Thus it undoubtedly helps to restore mobility, and to relieve pain in some cases of hallux rigidus and of coxa plana, instances in which we see a deformed joint the subject of a secondary arthritis. It is sometimes useful in the hypertrophic type of osteo-arthritis of the knee.

Arthroplasty for arthritis must still be considered as in the experimental stage. Its chief successes have been seen in the case of deformed or damaged joints, such as hallux valgus and old injuries of the elbow; in cases of bony ankylosis, and also in most of Putti's knee cases. It is sometimes successful in treatment of joints which are not subjected to through pressure, for example in the elbow and in the metatarso-phalangeal joint of the big toe. It is not so easy or so successful when applied to the major joints of the lower limb. The mechanics of the joint require careful study before an arthroplasty is undertaken. In a hinge joint, such as the elbow, it is not difficult to copy the original mechanics of movement, whereas in a lax joint, such as the shoulder, the result of an excision or arthroplasty is apt to be a useless arm. In general, the greatest objection to arthroplasty as a mode of treatment for arthritis, is that it may leave a fibrous ankylosis with a range of movement insufficient to serve any useful purpose but sufficient to cause pain.

Mr. Whitchurch Howell

mentioned three stages in arthritis, the pre-infective stage, the ankylosing stage, and the ankylosed stage. There were certain signs and symptoms which preceded the actual onset of an arthritis. The majority of arthritics were very thin, with little subcutaneous tissue and they suffered from poor circulation. They had acid saliva and acid sweat, and when these were made alkaline their physique improved and their arthritis, if it had begun, diminished, and in some cases disappeared. He (the speaker) also suggested that a number of advanced cases could be tackled surgically by a method of coaxing the joints one by one.

The most difficult cases were those in which there was painful arthritis of the shoulder-joint. The lower limb could be stabilized and so could the wrist, and an arthroplasty could be done at the elbow-joint; but with regard to the shoulder-joint, were they to stabilize it, or manipulate it, or what were they to do?

He described one case in which a young woman, after lying seven years in bed, recovered function in the hips, knees and wrists.

Mr. Zachary Cope.

The class of case in which very great deformities are a late consequence of polyarticular rheumatoid arthritis is very frequently regarded as hopeless, and such cases may drift to the infirmaries without any surgical attempt being made to better their condition. I have had several cases under my care in which the deformities were as great, or even greater, than those in the case related by Mr. Whitchurch Howell. In one case I straightened both knees by excision. In another case I excised successively both elbows and both knees with great benefit to the patient. I would call attention to the very great improvement in general health which results from the improvement in the condition of the joints ; weight is put on, and the patient becomes a much happier being. A point of pathological interest is the extreme atrophy of the cancellous tissue of the bones near the joint: the bone consists of a thin, brittle shell and a soft, fatty medulla. As a consequence, union after excision takes two or three times as long as in cases in which such atrophy has not occurred.

I would like to urge that very deformed joints be operated upon more frequently than hitherto.

Mr. Warren Low, C.B. (President)

said that twenty-seven years ago, during a very acute attack of dysentery (bacillary) in South Africa, he himself suffered from a multiple arthritis. With the exception of the fingers of one hand, all the joints of the extremities were involved, and for seven or eight months he was practically helpless. He remembered that he had a feeling that all the joint surfaces were extraordinarily soft and pulpy, and his instinct of self-preservation told him that he must not put any pressure on those surfaces. Further, he was not anxious to have them splinted, or in any way confined, as he felt there was danger of their becoming stiffened. In spite of temptations to do other things he stuck to this line of treatment, and at the end of eighteen months he was able to walk about with a certain amount of comfort. Surgeon-General Sir Alfred Keogh told him at the time that, of the dysenteric cases in the South African War, about 3 per cent. had suffered from this multiple arthritis.

Another officer who had a similar, but not quite such an extensive involvement at the same time as himself, during an attack of dysentery, was treated by his joints being put into plaster of Paris, with the result that they became very stiff, and when he (the speaker), himself quite recovered, saw this colleague some years later, he found him with stalactite masses at the elbows, and he had the melancholy satisfaction of excising one of his elbow-joints.

His own working hypothesis as to the pathology of these cases was that there were two factors at work—infection and mechanical injury—in very different proportions in different cases. In what might be called the rheumatoid condition it was mainly infection, in the monarticular variety probably the mechanical factor was a very large one, though he always felt that where there was marked infection there was apt to be an increased risk of the mechanical factor. For the joint surfaces, being softened, were more susceptible to injury from pressure, and this possibly explained why those gross changes were found in the lower more often than in the upper extremities.

Mr. Elmslie (in reply)

said that no doubt in the President's case fixation and immobilization would have led to a less satisfactory result than that which he had achieved by following out his own form of rest. But in another patient, less careful to protect his joints, advantage might be derived from putting a limb in plaster of Paris, and he cited one case in which mobility had been regained in a badly flexed knee-joint after this procedure. But in splinting, as in every other form of treatment, discrimination had to be employed in the treatment of the individual case.

Section of Therapeutics and Pharmacology.

President—Dr. GEORGE GRAHAM.

The Clinical and Pathological Effects of Hypnotic Drugs of the Barbituric Acid and Sulphonal Groups.

I.—Sir WILLIAM H. WILLCOX, K.C.I.E., C.B., C.M.G., M.D.

THE hypnotic drugs of the barbituric acid and sulphonal groups have for many years occupied my attention and interest. The opinions formed in regard to their use are based entirely on the actual experience and knowledge derived from clinical and toxicological cases that I have seen and studied.

These drugs have been dealt with by me in papers already published.[1]

The present paper is intended as a supplement to my previous communications, and represents additional experience in the clinical and pathological effects of the drugs in question.

I have for a long time been impressed by the potent therapeutic action of these drugs, either when given in a single large dose, or in doses repeated over a period of several days or longer. In cases that have come under my observation definite signs of organic paralyses of cranial and spinal nerves have occurred which pass away on discontinuance of the drug in cases where recovery occurs.

Marked mental symptoms accompanied by great depression, and sometimes by hallucinations, have been observed. The occurrence of these changes, which are identical with those of organic nerve disease, led me to believe that the taking of large doses or continued full therapeutic doses must cause definite organic changes in the central nervous system.

The late Sir Frederick Mott was very interested in the effects caused by hypnotic drugs of these groups, and in 1924, after one of the meetings at the College of Physicians, we had a long discussion on the subject.

Sir Frederick Mott decided to institute a scheme of research at the Research Laboratories at Hollymoor, Birmingham, in order to determine whether any actual pathological changes occurred in the central nervous system of animals after taking carefully regulated doses of the drugs in question. Dr. F. A. Pickworth and Mr. D. L. Woodhouse collaborated with Sir Frederick Mott in this research, and most striking and instructive results were obtained.

Sir Frederick Mott showed me in 1925 several of the microscopical specimens of the brain and spinal cord of animals which had undergone dosage with these hypnotic drugs. He had no doubt as to the occurrence of very marked changes in the central nervous system, and he told me that he was completely satisfied from results obtained by the most careful use of control animals, that the changes in question were the definite pathological effects of the drugs given.

I believe that this research is of far-reaching importance, and that it opens up a new line of investigation into the effects on the central nervous system caused by toxic substances, whether of artificial chemical nature or of biological origin, exogenous or endogenous.

It shows definitely that the clinical symptoms of paralysis of any part of the central nervous system following the use of therapeutic remedies cannot be regarded

1 "The Use and Abuse of Hypnotics" (Opening of Discussion at B.M.A. Meeting, 1913). "Veronal Poisoning" (Paper read at the International Congress of Medicine, 1913). "Drug Addiction" (Norman Kerr Memorial Lecture, 1923). *Brit. Med. Journ.*, 1923 (ii), 1013.

14 Willcox: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

as a mere temporary intoxication of harmless type. On the contrary, they are associated with definite objective changes in the central nervous system of a serious type.

Fortunately, many of the changes occurring may disappear after the discontinuance of the drug, a scientific confirmation of which has been observed clinically in human beings. Nevertheless, the results of this valuable research emphasize the supreme importance of so regulating the dosage of hypnotic drugs that toxic symptoms do not occur. For toxic symptoms mean organic changes in the central nervous system which may result in actual permanent damage to the nervous tissue.

From a study of my own cases and from a review of the literature which has been most carefully collated by Dr. Helen Young, I have no doubt whatever that the continued use of drugs of the barbituric acid and sulphonal groups leads to the formation of a definite addiction to the drug in question. The mental changes resulting from this drug addiction lead to serious moral changes, and suicide is most unfortunately one of the commonest of these.

The danger of the use of the hypnotic drugs has not, in my opinion, been sufficiently realized by the medical profession and the public, one reason for this being the absence of definite pathological evidence of the harmful effect on the central nervous system. Now that this evidence is forthcoming it is to be hoped that extreme care will be taken in the use and prescription of these hypnotic drugs.

Examples of drugs of the barbituric acid group are :—

Di-ethyl barbituric acid ...	(known as barbitone, malonal, hypnogen, veronal, etc., and its sodium salt medinal)
Proponal	(dipropyl barbituric acid)
Soneryl	(butyl-ethyl barbituric acid)
Dial	(di-allyl barbituric acid)
Luminal	(diphenyl barbituric acid) and its sodium salt (luminal sodium)

Recently a large number of barbituric acid compounds, derivatives and preparations have been placed on the market, among which may be mentioned the following :—

Allonal, a combination of ally lisopropyl barbituric acid with amidopyrin.
Veramon, a combination of veronal and ethyl aminophenyl dimethyl pyrazolon.
Gardenal, phenyl ethyl barbituric acid.
Cibalgin, a combination of dial and amidopyrin.
Phanodrom, cyclo hexemyl barbituric acid.
Beatol, a combination of veronal with extracts of valerian and jusquiame.
Somnifene, a mixture of veronal and allyl isopropyl barbituric acid.

The above list is by no means complete but it gives an idea of the rapidity with which these preparations of barbituric acid compounds are put on the market.

The addition of alkyl radicles of higher molecular weight than ethyl (C_2H_5) adds to the toxicity of the substance. Thus luminal, dial, proponal, gardenal, soneryl are more poisonous than veronal.

The fancy names conceal the composition, and unless minute scrutiny is made of the pamphlets relating to these drugs the barbituric acid nature will not be discovered. Also the glowing descriptions of their therapeutic action effectually conceal the fact that they may be very dangerous unless used with the greatest care.

The combination of a barbituric acid compound with an analgesic drug (e.g., allonal and veramon, etc.) appears to me to be dangerous, for whereas the latter may be taken repeatedly without danger of organic change in the central nervous system the former cannot be so taken.

In my opinion the greatest care is needed in the use of any compounds of barbituric acid, and the repeated administration of these preparations in one or more daily doses is undoubtedly dangerous.

The sulphonal group of hypnotics consists of :—

Sulphonal, which is dimethyl methane diethyl sulphonal.
Methyl sulphonal which is "trional."
Ethyl sulphonal which is "tetronal."

(I) Clinical Action.

The use of drugs of the barbituric acid and sulphonal groups is essentially for causing sleep. They are perhaps the most commonly used hypnotics. Until the introduction of veronal in 1902 the sulphonal group of drugs held a prominent position amongst the hypnotic drugs, but since that time the barbituric acid group of hypnotics has largely displaced the sulphonal compounds.

Barbituric Acid Compounds.—These are readily soluble in water. The therapeutic effect is rapidly induced and within an hour or so of the taking by mouth of a clinical dose drowsiness and natural sleep should ensue.

The drugs of this group in therapeutic doses act rapidly and are fairly certain hypnotics. It is for this reason that they have become so commonly used. A single therapeutic dose is usually followed by a satisfactory period of natural sleep, and no marked after-effects follow when the therapeutic action has passed away, which should be after six to twelve hours.

In cases of insomnia the use of hypnotic drugs is very apt to lead to the formation of the "hypnotic habit," and the patient looks forward to his hypnotic in much the same way as to his evening meal.

The danger in the case of the barbituric acid drugs is a very real one since it may lead to actual organic changes in the central nervous system. If these drugs are prescribed as hypnotics they should not be used more often than once or twice a week. At other times alternative methods of treatment of the insomnia are desirable.

Luminal, which in full doses (1½ to 5 gr.) is used as a hypnotic, has been recommended in the treatment of epilepsy in small doses (½ to 1 gr.) once or twice daily.

The barbituric acid group of drugs are essentially hypnotic in action and it is a mistake to prescribe them in frequently repeated doses for painful neuralgic conditions, since there is a risk of the occurrence of toxic symptoms. These drugs are primarily hypnotic in action, and other less toxic drugs, such as aspirin, phenacetin, pyramidon, are more safely used if analgesic effect is required.

The Sulphonal Group.—Sulphonal, trional and tetronal are powerfully acting hypnotics. They are very insoluble and are only slowly absorbed. In consequence of this the therapeutic effect of drowsiness and sleep may not commence for several hours, and then it will probably last for several hours. This delayed and uncertain effect has prevented this group of drugs coming into favour as hypnotics. They have been largely used in asylum practice. The same caution against repeated use applies to them with equal force as to the barbituric acid group of drugs.

(II) Toxic Action.

(1) *The Barbituric Acid Group.*

Acute Poisoning.—After the taking of a large dose, i.e., one larger than the maximum therapeutic dose, or in cases of idiosyncracy after the taking of a full therapeutic dose, headache, vertigo, and ataxy may occur in a few minutes, and within an hour or so the patient may fall into a deep sleep from which he can only be roused

with difficulty. This sleep may deepen into coma, and stertorous breathing which is sometimes Cheyne-Stokes in type may occur. The pulse is rapid and in the later stages of coma may become very feeble. Cyanosis occurs. The conjunctival reflex is lost in severe cases. The pupils are small and the reaction to light may be feeble or absent. The pupils are not pin-point as in the case of opium poisoning. The knee-jerks are absent and the plantar reflexes are usually either flexor in type or absent. In one case I observed an extensor plantar reflex.

Albuminuria of a mild type may occur and be associated with the presence of casts. There may be incontinence or retention of urine and incontinence of fæces. The comatose condition may last from a few hours to several days (see cases recorded in the Appendix, p. 37). In the severe comatose cases a very common event is the development of pneumonia. The temperature rises to 103° F. or so with rapid breathing and all the clinical physical signs of pneumonia. The pneumonia is broncho-pneumonic in type and usually takes the form of a massive broncho-pneumonia, large areas of lung becoming consolidated.

Sir Bernard Spilsbury and Dr. Newcomb have demonstrated by histological investigations the presence of broncho-pneumonia in a large percentage of the fatal cases of veronal poisoning at St. Mary's Hospital.

When pneumonia develops in a case of acute poisoning a fatal termination is almost always the result especially if this is accompanied by a feeble pulse. I have, however, known of recovery in one case of veronal pneumonia.

Fatal Dose.—Cases of death have been recorded after taking even such a small dose of veronal as 10 gr. though in such cases probably other factors were present and death was not entirely due to the drug. In a healthy adult it may be assumed that about 50 gr. represents an average minimum fatal dose. Recovery has taken place after much larger doses but gravely dangerous symptoms always occur in such cases.

Subacute and Chronic Poisoning.—This usually results from frequently repeated full therapeutic doses. An abnormal mental state may result, associated with marked depression and marked moral changes. Visual hallucinations sometimes occur and even delusions. Definite objective symptoms are present such as drowsiness, ptosis, squint and diplopia, nystagmus, facial weakness, etc. The speech may be thick and indistinct (anarthria). Difficulty of protrusion of the tongue and difficulty of swallowing have been observed. The gait is often markedly ataxic and may be reeling in type like that of alcoholic intoxication or of cerebellar disease. Tremors may occur. Skin rashes are sometimes present. They may be erythematous in type and of a rubeoliform or scarlatiniform character. Urticarial rashes and pruritus and œdema of the face have been observed. Slight amounts of albumin may be present in the urine. Hæmatoporphyrinuria has been observed but is much less common than in sulphonal poisoning. The above symptoms of subacute and chronic poisoning by the barbituric acid group of drugs may closely simulate such conditions as cerebellar disease, general paralysis of the insane, encephalitis lethargica, bulbar paralysis, and ataxic conditions of various kinds such as alcoholism, spinal cord disease (tabes dorsalis, ataxic paraplegia), peripheral neuritis, etc.

Veronal and its allies are quickly absorbed and rapidly excreted in the urine, and are not cumulative. The effects of continuously repeated doses, however, are cumulative; in other words, the objective nervous symptoms may remain for some days after a dose, and a repetition of doses may add to these symptoms which continue for some days or even weeks after all the drug has been excreted from the body.

Tolerance for the barbituric acid group of drugs does not in my experience appear to be established to any extent by long-continued dosage. In this respect these drugs differ from the alkaloidal addiction drugs such as morphine and cocaine.

The margin between the therapeutic dose and the toxic or poisonous dose is small. For this reason it is advisable to give, for hypnotic action, a dose well under the maximum therapeutic dose, e.g., 5 gr. of veronal or medinal at night is an adequate dose for an adult.

Professor Dixon has found by animal experiments that the margin between the full therapeutic dose and the lethal dose is quite small in the case of luminal.

(2) *The Sulphonal Group.*

Acute Poisoning.—The symptoms are similar to those of veronal poisoning except that the onset is much delayed owing to slow absorption. The comatose condition may last many days. Pneumonia of a broncho-pneumonic or hypostatic type may occur but it is less common than in the case of veronal poisoning.

Hæmatoporphyrinuria is a common symptom; albuminuria and casts may be present. The plantar reflexes were definitely extensor in the comatose condition of one case of fatal sulphonal poisoning under my care at St. Mary's Hospital.

Subacute and Chronic Poisoning.—The symptoms are similar to those of the barbituric acid group of poisons and all those described may occur in cases of the sulphonal group.

The slow absorption of the drugs causes the onset of the symptoms to be often much delayed, and their duration after cessation of administration of the drug may be more prolonged. In some cases these drugs may produce excitement and delirium. Skin rashes are much commoner than in the case of the barbitone group. They may be of erythematous or urticarial type and are often associated with marked prurigo. Vesicular and bullous eruptions and purpura have been observed.

Hæmatoporphyrinuria is a common but not a constant symptom.

Chronic Sulphonal Poisoning may closely simulate organic nervous diseases and toxæmias, exactly as in the case of the barbituric acid group of drugs.

Cumulative Action.—The slow absorption of these drugs renders them definitely cumulative in action, particularly where constipation is present. Thus the total dosage over several days may manifest its effects and lead to acute symptoms even when the daily dose has been within therapeutic limits.

Tolerance to the drugs is not developed by long-continued use.

Carelessness in Administration.—The slow action of these drugs has often led to the belief that they are of a harmless nature, and quite large doses have been daily administered to patients over long periods, when suddenly serious symptoms of poisoning have developed. It is important to realize that these drugs have a powerful toxic action on the central nervous system and cause quite definite organic changes in it. If used at all, the greatest care should be exercised.

Fatal Dose.—Death has occurred after one dose of thirty grains to a neurasthenic woman. Probably an average minimum fatal dose for an adult is about 75 gr., but recovery has occurred after very much larger doses. Several deaths have followed the daily use of from 10 to 20 gr. over periods of two months to twelve months.

Care is necessary in its use in mental institutions.

(III) Drug Addiction.

A definite addiction undoubtedly develops after the repeated daily use of these drugs and a marked craving results. I have observed this in many cases. The patients will use their utmost endeavours to obtain their drug and take it, quite regardless of any possible risk.

I cannot do better than quote as illustration of the veronal habit the statement of the mother at the inquest held on her son who had been a veronal addict for six years (H. E. T., 1912): "He took it first for pneumonia and never could give it up." "He told me he should never be able to give it up." "He seemed to lose all self-control when he wanted it."

As regards the sulphonal group, Dr. G. Hart (*American Journal of Medical Science*, 1901) says, "I have been impressed by the readiness with which patients acquire the trional habit and the difficulty of inducing them to abandon its use."

Addiction to the drugs of both of these groups leads to definite mental changes and marked depression, with alteration of character. The common termination of the continued addiction is suicide, usually brought about by the taking of a large overdose.

One patient (R. B., April, 1921), a young woman of 27, in well-to-do circumstances, had been taking veronal, 10 gr. daily for a year. She then drank, with suicidal intent and fatal result, 10 oz. of pure lysol. The veronal addiction and resulting depression was undoubtedly an important ætiological factor in this tragedy.

Addiction to the barbituric acid and sulphonal group of drugs differs from morphine and heroin addiction in that sudden discontinuance of the drug is not followed by severe withdrawal symptoms.

(IV) Post-mortem Changes.

The changes in the central nervous system are described by Dr. Pickworth. Cloudy swelling and degenerative changes occur in the cells of the liver and kidney, and definite degenerative changes in the heart muscle. The lungs show hypostatic congestion and œdema.

Well marked broncho-pneumonia, often of a massive type, is usually found in cases of the barbituric acid group and sometimes in those of the sulphonal group.

Barbituric Acid Group.

Toxicological Analysis.—The analytical details have been described in my paper on veronal poisoning. I should like to add to that some observations by the late Mr. John Webster who showed that where animal charcoal is used in the purification of the aqueous solution of the alcoholic extract much of the veronal is retained by the charcoal. In order to remove this the charcoal must be boiled with water and then again with pure alcohol in order to extract from it the absorbed veronal.

The distribution of the poison in fatal cases is characteristic. Since death rarely occurs under twenty-four hours the stomach and its contents only contain small quantities.

The liver, kidneys, brain, spleen, blood, and indeed all the tissues of the body, contain the poison in fairly even distribution. A remarkable feature is the peculiar eliminating power of the kidneys so that a relatively large proportion of the poison is present in the urine. This should always be obtained and reserved for analysis.

The liver does not appear to have the same power of picking up and retaining veronal as it possesses in the case of most other poisons such as arsenic and the alkaloidal poisons. The liver does not contain an appreciably higher percentage of the poison than the other organs, and the percentage is usually much lower than that found in the urine.

If an enormous dose of veronal be taken and death occurs in a few hours, which is most unusual, then large quantities may be found in the stomach contents as happened in one of the cases described.

Veronal was found by Mr. Webster in the cerebro-spinal fluid in one case under the care of Sir Bruce Bruce-Porter, which was seen by Sir James Purves-Stewart and myself.

In another case under the care of Sir James Purves-Stewart, which I saw, veronal was found in the cerebro-spinal fluid by Dr. Roche Lynch.

List of Toxicological Analyses

in seventeen coroner's cases—sixteen of these were made by the late Mr. John Webster, F.I.C., one by myself.

Statistics of fatal cases of veronal and barbituric acid group of poisons for England and Wales. Registrar-General's returns:—

VERONAL AND BARBITURIC ACID GROUP. FATAL POISONING. ENGLAND AND WALES.

Year	Male	Female	Total
1905	0	0	0
1906	0	1	1
1907	1	0	1
1908	4	0	4
1909	6	7	13
1910	9	6	15
1911	8	11	19
1912	12	4	16
1913	14	12	26
1914	12	18	30
1915	14	10	24
1916	5	5	10
1917	8	8	16
1918	6	6	12
1919	3	3	6
1920	3	6	9
1921	4	4	8
1922	3	3	6
1923	7	1	8
1924	4	4	8
1925	10	15	25
		Total ...	257

Drugs Placed under Part II Poisons Schedule 1913; under Part I Poisons Schedule 1918.

SULPHONAL GROUP. FATAL POISONING. ENGLAND AND WALES.

Year	Male	Female	Total
1905	4	2	6
1906	3	3	6
1907	3	1	4
1908	3	1	4
1909	3	2	5
1910	8	2	10
1911	2	0	2
1912	1	8	9
1913	0	1	1
1914	1	1	2
1915	2	1	3
1916	1	1	2
1917	1	1	2
1918	0	1	1
1919	0	0	0
1920	0	0	0
1921	0	0	0
1922	0	0	0
1923	0	0	0
1924	0	0	0
1925	2	0	2
		Total	59

Of the above, 9 cases were due to trional poisoning, 50 cases were due to sulphonal poisoning. Drugs of the sulphonal group were placed under Part II of the Poisons Schedule in 1913.

These statistics give some idea of the frequency of occurrence of cases of fatal poisoning, but there is no doubt that the actual number of cases exceeds these figures because:—

(1) Cases of death which are really due to veronal poisoning may be regarded as pneumonia or other condition producing coma.

(2) In the Registrar-General's Returns there are a number of cases each year classified as narcotic poisoning and an appreciable proportion of these are no doubt veronal poisoning.

(V) Poison Regulations.

Up to April 14, 1913, no restriction existed for the sale of drugs of these groups. On that date an order of the Privy Council placed them all under Part II of the Schedule of Poisons.

In 1918 in consequence of the frequency of fatal cases of veronal poisoning the barbituric acid group of drugs were by an order of the Privy Council placed in Part I of the Poisons Schedule. This restriction was followed by a definite reduction in the number of fatal cases from 1919 to 1924, but the 1925 figures showed a marked increase. It seems obvious that in the public interest some further restriction in the sale of these drugs is necessary. Regulations similar to those of the Dangerous Drugs Regulations as regards prescribing would undoubtedly be a most valuable safeguard.

(VI) Conclusions.

(1) The drugs of these groups have a powerful therapeutic action. In repeated doses they cause toxic symptoms of mental and physical type and definite organic changes in the central nervous system.

(2) Great care should be exercised in the use of these hypnotic drugs. They should only be supplied by prescription. The prescription should be marked "Not to be repeated, and to be retained by the Pharmacist." The total quantity of doses ordered should not exceed six, so that a risk of fatality may be avoided.

(3) The patient should be warned against the daily use of the drugs, and it would be a safeguard to avoid giving the drugs on consecutive days and to space the administration as much as possible.

(4) The medical profession should be seriously warned against the widely circulated advertisements and pamphlets relating to these groups of drugs, particularly those of the barbituric acid group. Such advertisements frequently recommend these drugs for conditions requiring repeated, continued administration without calling attention to the toxic effects which may result. They do not call attention to the very real danger of the development of addiction.

The continued introduction of these new modifications of barbituric acid derivatives—which must, necessarily, have had only an inadequate clinical trial—and the glowing advertisements which accompany them, are, in my opinion, seriously misleading to the medical profession and a grave danger to the public.

II.—F. A. Pickworth, M.B., B.S.Lond., A.I.C.

(1) Introduction.

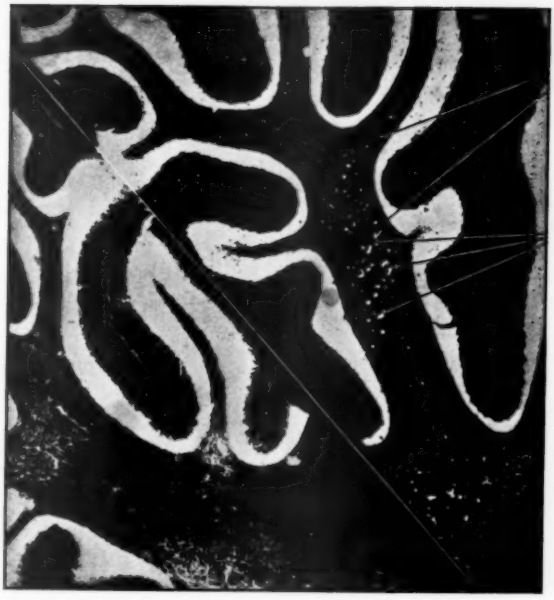
In the Norman Kerr Memorial Lecture on Drug Addiction [1] reference was made to the

> "probability of definite changes of a degenerative type occurring in the nerve cells of the brain, and that this pathological change leads to the formation of abnormal channels of transmission of nerve impulses whereby abnormal physical and mental effects are produced."

The occurrence of definite changes in the central nervous system of animals treated with hypnotic drugs has since then been demonstrated [2]. These changes, which regularly occur in chronic poisoning with any of the drugs of this series, consist of the deposition of a homogeneous mucinoid material in the white matter of the central nervous system (fig. 1). The mucinoid material has also been found actually within the nerve cells. The size of this material may vary from 5 microns to 60 microns, and in certain selected areas (near the dentate nucleus of the cerebellum) may occupy as much as 15 to 20 per cent. of the area seen in the

CEREBELLUM.

Veronal-fed monkey.

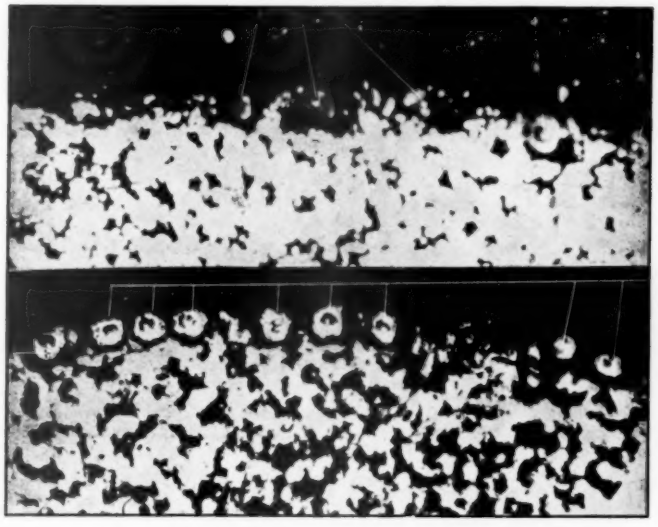


Normal control.

FIG. 1. Photograph showing mucinoid deposit in white matter of cerebellum.

CEREBELLUM.

Sulphonal-fed monkey.



Normal control.

FIG. 2.—Photograph showing disappearance of Purkinje cells and loss of Nissl staining substance.

PONTINE NERVE CELLS.

Veronal : Selected degenerated nerve cells. *Normal control.*

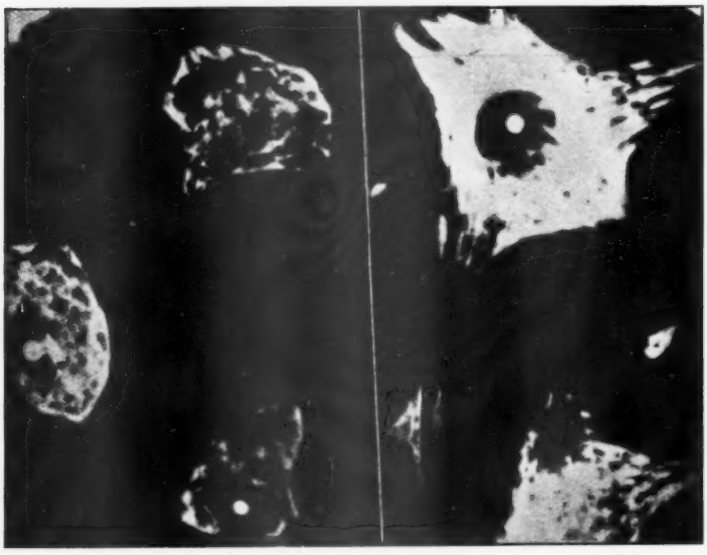


FIG. 3*a*.—Photograph showing disappearance of Nissl substance and cell degeneration.

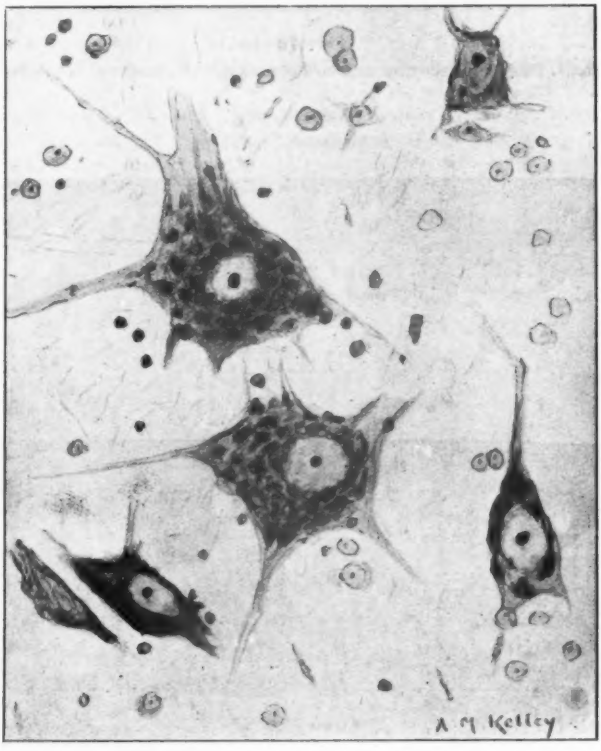


FIG. 3*b*.—Monkey, medulla. Normal control.

microscopic field. It stains metachromatically with toluidin and thionin, and when viewed through a Wratten G. screen is seen as a deep red substance with a blue background of tissue; it stains a deep golden brown with Bismarck brown. After formalin fixation it is insoluble in organic solvents and gives negative reactions for iron, fat, phosphorus, sugar, glycogen, or amyloid. In animals the treatment of which had been massive and prolonged, degenerative changes were found in the nerve cells. The Nissl substance was diffuse, diminished or absent (fig. 2). The cell outline was shrunken, irregular with loss of processes (fig. 3), and showed wrinkling

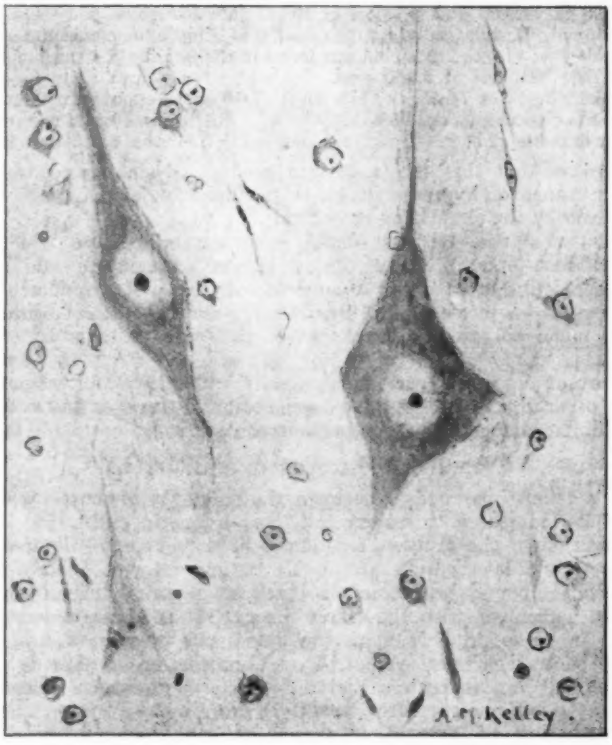


FIG. 3c.—Sulphonal-fed monkey. Loss of chromatin material and Nissl substance.

and abnormal staining reactions. The cells were occasionally surrounded by phagocytes. In some cases a marked eosinophil reaction of the nerve cells, especially of the Purkinje cells of the cerebellum, was present. In nearly every case there was a marked diminution of basophil staining substance which was evident in a naked eye comparison of sections stained together. All these experiments were carried out with identical but untreated animals, mostly from the same litter, for controls; these were reared together, killed by the same method, the tissue fixed, dehydrated, cleared and mounted together in the same block. Sections were of identical thickness and were stained together on the same slide.

24 Pickworth: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

The mucinoid material was deposited after treatment with each of the hypnotic drugs tested; these included drugs of widely different chemical composition:—(*a*) Sulphonal, (*b*) trional, (*c*) veronal, (*d*) soneryl, and (*e*) dial.

Summary of Treatment.

Cat (Ref. AS), weight 1,500 grm., fed with eleven daily doses of 2½ gr. sulphonal, and two daily doses of 5 gr. The animal showed continuous ataxia and anergia. Histologically, a large amount of dense compact spheres of mucinoid material in the white matter of the brain, cerebellum and cord.

Cat (Ref. MA) weight 2,500 grm., fed with six daily doses 2½ gr. of veronal, thirty-four daily doses of 5 gr. veronal and one dose of 10 gr. Marked ataxia and stupidity during the period of treatment. Histologically, a large quantity of compact mucinoid material in the brain cerebellum and cord; also the mucinoid was found in the interior of some ganglion cells.

Monkey (Ref. NI), weight 2,280 grm., fed with twenty-three daily doses of 5 gr. of veronal, two daily doses of 7½ gr., and two doses of 10 gr. Ataxia during the whole period; slept many hours on occasion, once for thirty hours. Histological examination showed a very large quantity of dense globules of mucinoid material in the brain, cerebellum and cord.

It is suggested that this substance is a product of neurone metabolism, which, under the influence of hypnotic drugs, is produced in large quantity and is less readily removed by the circulating body fluids.

A similar substance, but less dense, more crystalline and which stains less intensely, has been found in chronic disease [2], myxœdema [2], and in animals fed on vitamin-deficient diet [3]; also in animals poisoned with formic acid [4], and by various investigators in dementia præcox [4], and psychotic disorders; but no reference is made to possible treatment of the cases of this latter group with hypnotic drugs.

No important changes, apart from vascular dilatation and œdema, have been observed in other organs, except fatty degeneration of the liver and kidney, a certain amount of similar change being found also in the controls.

(2) Site of Action of the Drugs.

There is a definite parallelism between the solubility of most hypnotic drugs in lipides and fats (compared to water) and their hypnotic effect [5]. The probable physical condition of the drug *in vivo* must be considered:—Chloral hydrate as a pure drug is a notable exception to the Meyer and Overton theory, but it readily forms additive compounds with alcohols which are of an oily consistence, and therefore more in agreement with the above theory. It is generally accepted that the chief action of these drugs is upon the lipide and fat-rich tissues of the body, namely, the central nervous system [5]. Confirmation of this is given by the analytical findings in post-mortem material: one case of veronal poisoning [6] gave 0·68 grm. in the brain as compared with 0·18 grm. in the liver.

The distribution of veronal is similar in experimentally poisoned animals. Fredet-Fabrè [7] gave 1 grm. veronal to dogs (weight, 20 kilo.) and recovered 0·18 grm. from the brain and cord as compared with 0·04 grm. from the liver.

A pellicle of lipin-fat exists at the periphery of cell protoplasm, and a similar pellicle surrounds mitochondria [8], which are to be seen under dark-ground illumination by virtue of this refractive lipin-fat constituent. Much work has been done to show that all vital activity of cells is dependent upon the physio-chemical condition of these semi-permeable lipin-fat membranes.

Hypnotic drugs presumably dissolve in these membranes and alter their physio-chemical condition so that cell activity is hindered or inhibited. This conception of the action of hypnotic drugs necessitates that inhibition of vital activity shall occur in all tissues of the body including the blood, but predominantly in the central

nervous system; experimental research of other workers shows an action on the capillary wall, smooth muscle of uterus, ureters and intestine; which will be referred to later.

From the work of Magnus [9] who found that certain reflexes are lost in a regular sequence—the first noticed action being alteration of the respiration rate—it is extremely probable that these hypnotic drugs act directly on nerve fibres inhibiting conduction (Adrian [10] has shown that volatile narcotic drugs, such as ether and chloroform, have a strictly local action, preventing the conduction of nervous impulses along the fibres); this would account for the sequence of inhibition of reflexes, the long accessible nerve tracts, e.g., the phrenic, being soonest affected.

It must be emphasized that the effects of these lipide-soluble drugs are never instantaneous; it takes some considerable time for pure chemicals to diffuse from one solution into another immiscible solvent, and, similarly, hypnotic drugs are distributed only slowly in the body. The peripheral tissues are affected first, whilst several hours are required for the drug to reach the more central parts of the nervous system; also the quantity of drug dissolved by the tissue elements is usually very small owing to the fact that these drugs are also to some extent soluble in water and serum. Even with enormous single doses of the drug animals will live many hours, and a woman who took twenty 10 gr. tablets of dial survived three days [11]. Definite histological changes due to complete or partial disruption of cell membranes are seen only in the later stages or in chronic poisoning with the drugs.

(3) Hypnotic Action.

It has been found that it is difficult not to fall asleep [12] under successful voluntary muscular relaxation and inhibition of mental effort, which is possible under certain conditions of quiet or regular monotonous sounds; and when the sense of weight due to gravity is minimal, as in a comfortable armchair. This means that a very important factor in the production of sleep consists in the inhibition of all afferent peripheral stimuli except those which can be utilized in automatic reflex acts, such as breathing, etc. The prevention of conduction of nerve impulses along nerve fibres affords a simple explanation of the hypnotic action of this series of drugs. In addition there is the reduction of vital activity of the nerve cells themselves by the action of the drug upon their semipermeable membranes.

The consequence of loss of reflexes is quite constant; in rabbits there is first, slowing of respiration, then disturbances of the following: sitting posture, corrections of abnormal positions, running, righting reflexes, reactions to progressive movements, etc., and the depth of narcosis can be estimated by the presence or absence of these reflexes [9]. The resemblance between the hypnotic action of these drugs and normal sleep is only superficial. In cats the margin between sleep and fatal coma is quite narrow, although a greater latitude occurs in monkeys [2]. The lethargy of veronal poisoning may be more acute even than that of encephalitis lethargica. In one case of veronal poisoning lethargy lasting thirteen days occurred [13]. If the vital activity of all the body cells is inhibited natural anabolic repair processes cannot proceed in the same manner as occurs in normal sleep. This is borne out by the abnormal irritability and anergia which often ensue on awaking from the effects of the drug. The hypnotic effect is ascribed by many to the direct action of the drug upon the vessels of the brain causing dilatation and lowered oxidation [14], [15]; Cushny denies this and states that the dilatation of the vessels is secondary [5].

(4) Effect upon the Vascular System.

Contradictory statements appear in the literature with regard to the action of hypnotic drugs upon the vessels, but most are agreed that with moderate or low concentrations of the drugs a dilatation occurs. Dilatation of the capillaries is

regularly observed with veronal in concentration of 0·1 per cent. [14, 16], and this has been called the "true action of all barbital derivatives" [16].

Perfusion of an excised brain with veronal causes an increase in the flow due to dilatation of the cerebral vessels [16]. Administration of the drug causes a vasodilatation with increase of the blood-flow, and there is slowing of the pulse [14] and a fall in temperature. It must be carefully observed, however, that in spite of this increased blood-flow the tissue respiration is much below normal, due partly to the difficulty of respiratory exchange and partly to the disturbance of the normal alternative activity and the rest of the groups of capillaries; this is seen in the frequent occurrence of cyanosis. In fatal cases post-mortem examination shows a general hyperæmia of all organs with the heart arrested in systole, the right ventricle being dilated and full of blood [15].

It is stated that the drugs act directly upon the capillary vessel wall and not upon the muscle of the arterioles [14], because it is found that vasoconstriction may occur upon suitable stimulation, and therefore vasomotor nerve terminations and sympathetic ganglia are not entirely paralysed [14]. But other workers have found a definite loss of tone of the smooth muscle of the intestine, ureters and uterus by veronal in concentrations even as low as 1 in 2,000 to 1 in 100,000; much larger concentrations prevent the peristaltic movements of the intestine [17].

It has been demonstrated that the slowing of the pulse and increase of blood-flow in frogs due to veronal is much less if the animal's brain is first excised [14]; the figures given show that veronal produces a greater amount of slowing of the pulse and increase in blood-flow in animals with intact nervous system than ten times the amount of drug in a brainless animal. Contrary to the opinion of the authors (who uphold the vascular action of the drug as primary) the figures seem to confirm positively the essential action of the drugs as upon the central nervous system. The vasodilatation, diminished blood-pressure, slowing of pulse-rate and lowered vital activity of the tissues may possibly result in the accumulation of certain histamine-like substances which occur in normal active tissue [18] and which continue, as a vicious circle, the vascular effects; this may be of importance in the understanding of certain undesirable late sequelæ.

(5) Effects upon the Central Nervous System.

In addition to the depression of vital activity of the nerve cells following alteration of the physio-chemical condition of the semi-permeable membranes, and the probable local inhibitions of conduction of nerve impulse along the nerve fibres which are responsible for the immediate effects, there is, in chronic and subacute poisoning, the added effect of the presence of the mucinoid material (or its precursor) in the substance of the nervous tissue. There are three possible results, which deserve consideration, due to its presence. First, the local pressure effects upon nerve fibres, causing displacement, distortion and compression (fig. 4); the combined action of groups of deposit of mucinoid leads to the opinion that there may be an analogy between certain symptoms common in veronal poisoning and disseminated sclerosis [2, 11]. Secondly, the quantity of the deposit, at least in experimental animals, is considerable and possibly influences intracranial circulation. Thirdly, if, as we believe, this mucinoid is a metabolic product, its presence, according to "mass action" effect, must inhibit forward metabolism (tissue recovery and repair processes).

The cumulative effect of these drugs is well known [19].

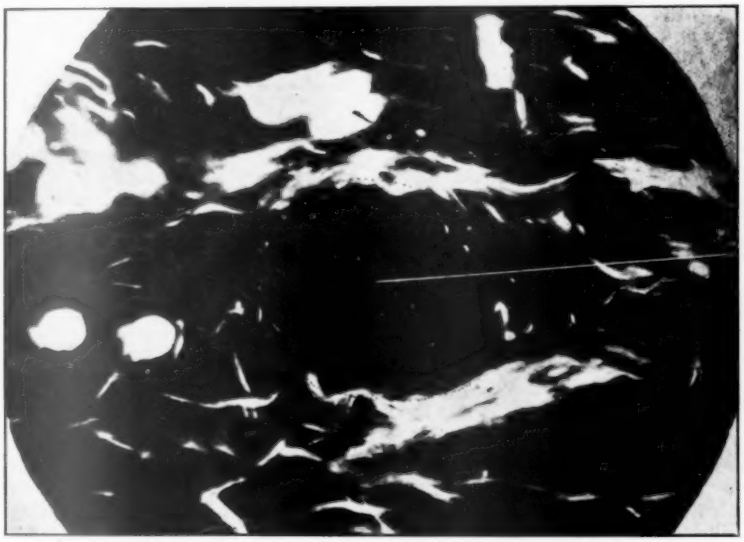
We have found both cumulative action and a certain degree of tolerance in animals—one of a pair of cats previously had treatment with veronal; both were given 10 gr. of veronal; the cat previously treated showed effects earlier and also recovered from the major symptoms quicker than the other.

Lipide-soluble drugs introduced into the system readily affect the peripheral system, which, in turn, recovers comparatively quickly as excretion takes place; central parts of the nervous system are more slowly disturbed, requiring prolonged treatment with drugs to produce marked effect; the excretion is very slow owing to the protected position and partially drug-saturated lipides in the neighbourhood. The relation of this process to the orderly sequence of loss of reflexes and their recovery, previously referred to [9], will be easily seen.

In poisoning by drugs of this series, especially in the later stages, nervous affections are a predominating feature. Many of these, fortunately, are transient, and clinical recovery is the rule (in this connexion we have shown that the mucinoid deposit slowly disappears from the nervous system upon cessation of the drug [2]).

CEREBELLUM.

Veronal-fed cat: Thionin blue stain.



A large spherular mass of mucinoid material.

FIG. 4.—Photograph showing nerve fibre distorted and pressed together by the mucinoid material.

However, in spite of the fact that drugs of this series may be taken for a long time without apparent gross harm, many cases suddenly show undesirable symptoms which progress, in spite of treatment, to a fatal issue [20, 21, 22].

Amongst the common clinical nervous symptoms is the disappearance of reflexes and the occurrence of an extensor plantar response (the latter ascribed by one observer as secondary to liver insufficiency [23]). Polyneuritis has been described [22, 24, 25]. Ocular paralyses are common [13, 26]. Various paralyses of sensation with severe pain or analgesia have been noted [27]. Fatal acute ascending paralysis is described by Stockton [28].

Cerebellar symptoms are usually well marked.—Ataxia, dysmetria, nystagmus, and, in one case, a tendency to fall backwards [29]. The occurrence of extreme

hypotonia of muscles has been emphasized by Achard [11], the same observer also noting a "doll's eye" reaction to position of body as sometimes seen in encephalitis lethargica. Speech is often affected, may be scanning, as in disseminated sclerosis [11], with omission of syllables; frequently rambling and incoherent. Memory, especially for recent events, is often faulty. Perception and association are retarded; there is emotional instability, restlessness, insomnia, and mental deterioration [15, 31].

Acute cases often show a period of delirium [30] or of loquacity and gesticulation [11]; hallucinations and delusions have been described [22]. Definite psychotic symptoms may occur [30]. Euphoria or melancholia with suicidal intent [11] and a case of epileptiform fits after sulphonal are recorded [31]. Subacute poisoning may simulate uræmic coma, cerebral tumour, general paralysis or encephalitis lethargica [13, 11, 15], the last reference showing that the diagnosis of encephalitis lethargica was made before a history of veronal poisoning was obtained.

(6) Other Symptoms.

Rashes occur in 20 per cent. of veronal takers. One woman had a rash for three years, which disappeared on cessation of the drug [32]. Five per cent. of luminal takers develop dermatoses [33].

Hæmatoporphyrinuria sometimes occurs with drugs of the sulphonal series and may be accompanied by epigastric pain, vomiting, ataxia, confusion, and collapse leading to death [5, 20, 24]. One case is recorded of hæmatoporphyrinuria due to veronal poisoning [34]. *Toxic nephritis* due to sulphonal is described [35] and may be of importance, since excretion is delayed in cardio-renal affections.

Pulmonary œdema is regularly observed in fatal cases and *broncho-pneumonia* commonly occurs "due to the depressant action of the drug upon the neurotrophic system rendering the pneumonic tissue much more susceptible to bacteria" [6, 15]. *Hæmorrhage* may be found in the gastric mucosa; and in animals this may occur even when veronal is given subcutaneously [14].

Many observers agree that the continued use of the drug has a deleterious effect upon the general health; we ourselves noticed that quite small daily doses of veronal caused a lively, clean and contented cat to become sullen, miserable and unkempt.

(7) Toxicology.

Veronal is fatal in doses of 1½ grm. per kilo. for frog, 0·4 grm. per kilo. for rabbits, 0·35 per kilo. for cats [14]. The minimum average fatal dose for man is 4·5 grm. [36], or 50 gr. [37]. Death has occurred with 15 gr. [37] and recovery with 125 gr. [15]. Cases are on record in which large amounts have been taken over a long time without obvious gross harm; one woman took 6 lb. of sulphonal over a period of twelve years [21], but fatal results often occur suddenly after continued use of the drug in moderate doses, e.g., 20 gr. sulphonal daily for three months [20].

The drug is excreted solely by the kidneys, 90 per cent. is recoverable in the urine when small amounts are given; with large amounts a retention of the drug in the tissues occurs and only 50 per cent. is recoverable [15].

Post-mortem examination shows general hyperæmia of all organs, hæmorrhages of the stomach, pulmonary œdema, broncho-pneumonia, and the heart arrested in systole with right ventricle dilated and full of blood. Fatty degeneration of the liver and degeneration of kidney tubules are also observed [15].

The drug is recoverable from the brain and liver [6], and has been found in the cerebro-spinal fluid from a case of dial poisoning [11].

A summary of the histological examination of the tissues in a fatal case recorded by Huseman [38] is as follows:—

Fatty changes in heart muscle; vessels and endothelium laden with fat, liver shows fatty changes chiefly at central parts of acini; Kupffer cells full of fat. Kidney shows little fat (endothelium only) but degeneration of tubules. Central nervous system shows a diminished affinity for methylene blue; ganglion cells show fine fatty droplets chiefly near the nucleus; Nissl substance in fine dust; cell outline altered, swollen or wavy; increase in glia cells, especially in the cornu ammonis; collection of metachromatic substance around vessels and cells of the basal ganglia and cortex.

The distribution in the various organs is given by Fredet-Fabre as follows [7]: 1 grm. veronal given to 20-kilo. dog.

130 grm.	brain and cord.	Veronal found	= 0·180	90 grm.	kidney.	Veronal found	...	= 0·081
420 ,,	blood	,, ,,	= 0·043	400 ,,	muscle	,, ,,	...	= 0·055
810 ,,	liver	,, ,,	= 0·038	110 ,,	fat	,, ,,	...	= 0·016
40 ,,	urine	,, ,,	= 0·023	50 ,,	spleen	,, ,,	...	= 0·022

The paralytic effects of veronal in animals in small doses are almost completely antagonized by pyramidon (39·9), and to a less extent by antipyrine (39).

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HISTORY.

In the history of medicine there are repeatedly obscure allusions to substances used for the purpose of hypnosis, but from 1854 to 1857 the soporifics included only the four groups:—

(1) The spiritus containing alcohol, ether and chloroform.
(2) The opiate, monopolized by opium.
(3) The Indian hemp group, containing hemp and its many preparations, and
(4) The solanaceous group, containing belladonna, henbane and stramonium.

Besides these there were drugs of less importance, such as lettuce and hop. It was, however, not until twenty years after the introduction of the general anæsthetics, in the latter half of the 19th century, that the attention of investigators was diverted to the quest for hypnotics, with the result that many of these drugs have been added to therapeutics.

30 Young: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

Beginning with the examination of chloral hydrate by Liebreich, in 1869, the employment of other groups followed, and of these the drugs of the barbituric and sulphonal groups have been widely used.

Sulphonal, discovered by Baumann, and introduced into medical practice in the year 1888, has had an eventful history, which may conveniently be divided into three periods.

During the first period the drug seemed to approach the ideal as a hypnotic, and, due principally to its property for calming the motor excitement of the insane, its praises were sung at meetings of the medico-psychological societies, and it rapidly gained favour with both physicians and patients.

The second period was one of disillusionment, and, later, distrust, during which contributions to the literature on sulphonal contained unfavourable reports, beginning in 1889 with Griffith's "Remarks on the Unpleasant Effects of Sulphonal," and continuing with cases of both acute and chronic, and sometimes fatal, poisoning. It is stated that before 1894 [1], that is, within six years of its advent, sulphonal had been responsible for eighteen deaths, most of them medicinal or accidental, while by 1900 [2] a further sixteen fatal cases had been added to this list. From 1900 to 1909 about eighteen authors recorded many more cases of acute and chronic sulphonal poisoning, some terminating fatally, and some in permanent dementia [3]. And so in 1909, in reference to sulphonal and its allies, we read that "trional is the best of the three, sulphonal should be dropped, tetronal is too dangerous" [4].

Again, in 1910, at a meeting of the Scottish Division of the Medico-Psychological Association, the superintendent of a mental hospital stated that "the true death-roll from sulphonal was very much greater than that recorded, as both the acute form of poisoning by sudden collapse, and the more chronic form with hæmatoporphyrin in the urine, may be mistaken for other conditions [5].

In addition, although death may occur in a few hours, or days, or after months, it has also been known to occur some time after the withdrawal of the drug. There were, moreover, in 1910, at least ten medical superintendents of asylums in Great Britain who had abandoned completely the use of sulphonal, and in a larger number of asylums the drug was used very seldom and then only in small doses and for short periods, and it was withheld in all recent cases of insanity in which there was hope of recovery.

However, this disuse of sulphonal was not in all these asylums due entirely to its dangers, but partly to the discovery of other synthetic drugs, such as trional and veronal, and partly to the fact that the routine treatment of acute cases of insanity with sedatives was less in vogue than in earlier years [7].

During *the third period* of its history sulphonal became partially reinstated in favour, and at the end of the year 1910 we read that "sulphonal with its two ethyl groups is still the most popular, and trional is more popular than tetronal, which is seldom employed" [8]. By 1913 sulphonal was again used by the majority of asylum superintendents and, being better understood, fatal accidents seldom occurred, although cases of poisoning, with unpleasant, and even grave symptoms, were from time to time reported [9].

In 1916, in England, two deaths, one accidental and one suicidal, from sulphonal, were registered [10], and in the year 1920 a case of sulphonal poisoning, terminating fatally, was recorded.

In the "British Pharmaceutical Codex," 1923, we find the statement that, due to its cumulative action, and tendency to destroy hæmoglobin in the blood, with the production of hæmatoporphyrin in the urine, sulphonal, for any but occasional use, is one of the most dangerous hypnotics.

Trional.

Methylsulphonal, introduced soon after sulphonal, partially displaced it in favour, and, being sold under the trade name of trional, it unfortunately produced the same type of poisoning.

During the years 1893 to 1913 there were recorded more than seventy cases of trional poisoning, both acute and chronic, about thirty of which were fatal. It appears that trional afterwards fell into disuse, and during the years of the war, 1914-1918, we find recorded only one acute case of fatal trional poisoning, and two cases of chronic poisoning, one of which was fatal.

Tetronal.

Tetronal, a compound similar in both chemical and physiological properties to sulphonal, and introduced about the same time as trional, has been responsible for cases of fatal poisoning, but its use in medical practice has been very much limited.

Barbituric Group.

From the sulphonal group we turn to the barbituric group of drugs, derived from urea, of which barbitone (di-ethylbarbituric acid) has been most widely used. Formerly described in the " Codex " under malourea, and sold under the trade names of veronal, malonal and hypnogen, it was introduced into therapeutics in 1902 and was followed by medinal (sodium veronal), in the same year; the latter soon became a popular hypnotic.

To these drugs were added, in 1906, dial (diallyl-barbituric acid) and luminal (phenyl-ethyl-barbituric acid) with its salt, sodium luminal. The last two have been used in the treatment of epilepsy and the various forms of functional neurosis, while veronal, medinal and dial, and later soneryl (butyl-ethyl barbituric acid) and somnifene (isopropyl-propenylbarbiturate of diethylamine) with others less well known of the group, have been chiefly prescribed for the induction of sleep.

Exhaustively studied by Willcox in England [11] and later, clinically, by Tardieu in France, the toxicity of veronal is now widely known, but, due to the variety of names of this substance, of its allies, and of their derivatives, patients who have learned by experience to abandon the drug known by one name, often take it unwittingly under another. Cases both of *acute veronal poisoning* and of habit [12] formation began early to be reported, and as " eight grains of veronal require three or four days to excrete " [13] sudden symptoms of poisoning, from its regular use, have often occurred.

Veronal habit has been recorded, due in part to the fact that a feeling of spurious well-being often follows its use, and that the conditions for which it is given are those that recur. From a careful survey of case records of poisoning from drugs of this group, insomnia and types of recurrent headache stand first, and the treatment of alcoholism next, as a cause for their use. But it is stated " the terrors of insomnia are incomparable to those of drug addiction which leads to an unstable condition of the nervous system," thus fixing the habit [14].

Suicides as a result of the veronal habit are not infrequently recorded, and it is stated that deaths have been more from veronal in one year than from patent medicines in a century [15], while in England and Wales in the years 1911-1913 veronal occupied the seventh place as the cause of death from all poisons [16].

Again, there are recorded cases of *idiosyncrasy* to even small doses of drugs of this group [17], but the majority of cases of poisoning following small single doses have been reported either in elderly patients [18] or in those cases in which other factors were present [19].

32 Young: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

In England the use by the public of veronal is said to have superseded the use of sulphonal, when the latter was placed in Part II of the Dangerous Drugs Schedule, and the increasing number of deaths from taking veronal led to its being placed, in its turn, on the Poisons Schedule (I) [20]. From 1906 to 1925 inclusive, 253 deaths from poisoning by drugs of the barbituric acid group have been recorded in the Registrar-General's statistics for England and Wales. In 1925 there were twenty-two deaths.

In America, in *one* hospital, during two years, sixty-one cases of barbital poisoning were admitted, several of which were fatal. Forty patients of this series were under 40 years of age, and the youngest, a girl of 17, had been medicinally treated with barbital. Eighteen of these patients were veronal addicts, and nineteen had taken veronal with suicidal intent, while several, on recovering from veronal poisoning, attempted suicide by other means [21].

In Germany the following figures point to the increase in the cases recorded of veronal poisoning admitted to one hospital (Stuttgart) [22] from the years 1907 to 1921:—Years 1907-1911, one case in hospital; 1911-1920, average of one case a year in hospital; 1919, four cases in hospital, all suicides; 1920, three cases in hospital, all suicides; 1921 (January to July) three (female) cases of suicidal intent. Of these sixteen cases five were fatal. In Hamburg, between the years 1904 and 1913, forty-five cases of veronal, taken with suicidal intent, are recorded. In Berlin, there were twelve fatal cases of veronal poisoning in the years 1905 to 1907 [23].

The total number of cases recorded in the literature, including those for the year 1926, are over 400, of which approximately one-third were fatal, and as only a small proportion of cases of veronal poisoning are recorded the actual number must greatly exceed these figures.

In conclusion, two fatal cases of poisoning by drugs of this group are of interest on account of their differential diagnoses.

Case I.—Miss X. Aged 40. She suffered from chronic infection of both antra following operations on the nasal septum for the relief of asthma, but was otherwise strong physically, and was normal mentally. The toxæmia from focal infection caused insomnia and restlessness, and for four years veronal was prescribed, in various countries, for the relief of insomnia.

Twelve months before her death the following symptoms appeared in this order:—

(1) Increasing restlessness and insomnia.
(2) Mental worry.
(3) Thick, indistinct, slurred speech.
(4) Urticarial eruption of skin with itching of skin, especially of the legs.
(5) Headaches and many other subjective symptoms, e.g., that "head was made of cotton wool" and was "buzzing round inside."
(6) Vertigo with feeling that stationary objects were moving round her.
(7) Nausea.
(8) Irritability and depression.
(9) Diplopia and indistinct vision, following larger doses.
(10) Ataxic gait with dragging of the feet and difficulty in walking upstairs. At times reeling and staggering gait so that the patient walked with the aid of two sticks.
(11) Loss of insight into condition, patient knew the symptoms of veronalism, but could not connect these with her own illness. Taking veronal gave her a sense of well-being and temporary relief from restlessness and sleeplessness, and she could not give it up.
(12) Conjunctivitis and inflammation of eyelids.

In April, 1925, patient entered a nursing home in London, and was prescribed medinal gr. x and aspirin gr. x taken at night, for the relief of headache, pain in the left eye and insomnia. After the dose had been given the patient took her own dose of somnifene which had been prescribed abroad in place of veronal several weeks previously. (20 to 30 minims somnifene = veronal gr. v.)

Next morning she was found to be comatose; stertorous and Cheyne-Stokes breathing, raised temperature, feeble pulse and cyanosis occurred, the symptoms arousing the suspicion of a cerebral lesion. Previous to the onset of the acute symptoms the condition had been treated as post-influenzal functional neurosis.

Section of Therapeutics and Pharmacology 33

Pus from both antra discharged down the nostrils shortly before death, which occurred about twenty-six hours after medinal had been given. The time at which patient took somnifène is not known.

Post-mortem showed no organic lesion, but signs of pneumonia. Analysis showed :—

In the liver	2·5 gr.	veronal
„ urine	7·8 „	„
„ stomach and contents ...	0·8 „	„
„ kidneys	1·0 „	„
Total ...	12·1 „	„

Estimated total dose taken 50 to 100 gr. veronal.

Case II.—In 1924 a young waiter (a total abstainer) in an Edinburgh private hotel, suddenly became excited, and was taken in a taxicab to Edinburgh Royal Infirmary. In the taxicab he became unconscious, and on admission was comatose, and later was found to be suffering from all the acute symptoms of poisoning in the case given above. He died within twenty-four hours of admission, and at the post-mortem the organs were found to be perfectly healthy, except for signs, in the lungs, of hypostatic pneumonia. The provisional diagnosis had been cerebral abscess, and the death was certified as pneumonia.

Many weeks later there was indirect evidence that an American visitor to the hotel had given barbitone to the patient for the relief of neuralgia.

These cases suggest that, if, even as late as two and three years ago, veronal poisoning (both chronic, lasting many months, and acute) was difficult to diagnose, in earlier years numerous instances had not been detected.

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[1] FRIEDLÄNDER, *Therap. Monatsh.*, 1894, viii, 232 (Thirty-four Fatal Cases). [2] TAYLER and SAILER, *Contributions from William Pepper Laboratory of Clin. Med.*, 1900, 124. [3] ROBERTSON, G. M., *Journ. Ment. Sci.*, 1911, lvii, 286. [4] *Brit. Med. Journ.*, 1909, i, 555. [5] ROBERTSON, G. M., *Journ. Ment. Sci.*, 1911, lvii, 283. [6] SMITH, SIDNEY, "Forensic Medicine," 1925, "Sulphonal." [7] ROBERTSON, G. M., *Journ. Ment. Sci.*, 1911, 275. [8] *Brit. Med. Journ.*, 1910 (ii), 1113. [9] ROBERTSON G. M., *Journ. Ment. Sci.*, 1911, lvii, 276. [10] Registrar-General's Statistics, 1916. [11] WILLCOX, *Lancet*, October 25, 1913, 1178. [12] LAUDENHEIMER, *Therap. d. Gegenw.*, 1904, vi, 47. [13] "British Codex," 1923, 176. [14] WILLCOX, "Drug Addiction," The Tenth Norman Kerr Memorial Lecture, 1923, *Brit. Journ. Inebriety*, January, 1924. [15] PARRY, P. M. C. E., *C. D.*, 1 13, 600. [16] PETERSON, HAINES and WEBSTER, "Legal Medicine and Toxicology," 1923, ii, 660. [17] WILLCOX, "The Use and Abuse of Hypnotics," *Brit. Med. Journ.*, September 13, 1913, 661. [18] POLLITZER, "Veronal Poisoning," *Journ. Cutan. Dis., including Syph.*, N.Y., 1912, xxx, 185. [19] WILLCOX, *Lancet*, 1913 (ii), 734, 1178. [20] WILLCOX, "Drug Addiction," *Brit. Journ. Inebriety*, January, 1924, 22. [21] LEAKE and WARE, *Journ. Amer. Med. Assoc.*, 1925, lxxxiv, 434. [22] BOENHEIM, *Berl. med. Klin.*, 1921, xvii, 1263, also SIEVEKING, *Viertjahrschr. f. ger. Med.*, 1918, lvi, 163. [23] JACOBI, ("Concerning Cases recorded to end of 1907, of Death by Veronal Poisoning.") *Vierteljahrschr. f. ger. Med.*, 1909, xxxvii, 234.

INDEX.

Veronal.

34 Young: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

Year	Cases	Fatal	Authors	References
1905	1	0	Geiringer	*Wien. klin. Woch.*, xviii, 1243.
—	1	1	Harnack	*München. med. Woch.*, lii, 2269.
—	1	?	Kuhn	*Hosp. Tid.*, Kobenh. 4 R, xiii, 32.
—	1	1	Lewinski	*Zahnärztl. Rundschau*, Berl., xiv, 837.
—	3	1	Alter	*München. med. Woch.*, lii, 514.
—	1	1	Wills	*Brit. Med. Journ.* Epitome, November 4.
1906	1	0	Wills	*Brit. Med. Journ.* (i), 498.
—	?	?	Hampke	*Inaug. Diss.*, Leipzig.
—	1	1	Anon.	*Boston Med. and Surg. Journ.*, cliv, 113. "Death, veronal."
—	1	1	Germann	*Journ. Amer. Med. Assoc.*, xlvi, 1999.
—	1	0	Mörchen	*München. med. Woch.*, liii, 1231.
—	3	2	Umber	*Med. klin.*, Berl., ii, 1254-1256, also *München. med. Woch.*, liii, 2556.
—	3	2	Zörnlaib	*Wien. med. Woch.*, lvi, 2454.
1907	1	0	Bulkley	*Journ. Amer. Med. Assoc.*, xlviii, 1864.
—	1	0	House	*Journ. Amer. Med. Assoc.*, xlviii, 1348.
—	1	0	Woolley	*Journ. Amer. Med. Assoc.*, xlix, 2153.
—	2	0	Zengerly	*Strassb. med. Ztg.*, iv, 45.
—	1	0	Gwozdecki	*Lwow. Tygodn. Lek.*, ii, 221-223.
—	4	3	Nienhaus	*Cor. Bl. f. Schweiz. Aerzte*, Basel, xxxvii, 336-341.
—	1	0	Papp	*Gynogyaszat*, Budapest, xlvii, 484.
—	?	?	Stucky	*Louisville Monthly Journ. Med. and Surg.*, 1907-08, xiv, 241-246.
—	1	0	Lyons	*Brit. Med. Journ.* (i), 259.
1908	12	0	Wolters	*Med. klin.*, Berl., iv, 182.
—	1	0	Clerk	*Journ. Amer. Med. Assoc.*, li, 1229.
—	1	1	Parsons	*Brit. Med. Journ.* (ii), 832.
	And child	And child		
—	1	1	Koch	*Nederl. Tijdschr. v. Gen. Amst.*, 2 R, xliv, 1 afd. 326.
—	4	0	Neumann	*Berl. klin. Woch.*, xlv, 1682-1686.
—	9	9	Panzer	*Vrtljschr. f. ger. Med.*, Berl., xxxii, 311.
—	4	0	Steintz	*Therap. d. Geg.*, Berl., xlix, 203-211.
1909	1	1	Davies	*Brit. Med. Journ.* (ii), 1154.
—	2	2	Anon.	*Lancet* (ii), 760. "Deaths, veronal."
—	1	0	Eckel	*N.Y. Med. Journ.*, xc, 118.
—	1	0	Iwanow	*Russ. med. Rundschau*, Berl., vii, 595-607.
—	12	12	Jacobi	*Viertschrift f. ger. Med.*, Berl., 3 F, xxxvii. 222.
—	?	?	Van Engelen	*Ann. Soc. de Méd. lég. de Belg.*, Brux., xx, 39-44, Discussion 9.
—	1	1	Walker	*Lancet* (i), 1557.
1910	?	?	Margerie	*Inaug. Diss.*, Erlangen.
—	1	0	H. (W. M.)	*Brit. Med. Journ.* (i), 552.
—	?	?	Ide	*Rev. méd. de Louvain*, 78-80.
—	1	0	Martin	*Brit. Med. Journ.* (ii), 457.
—	5	0	Sowden	*Brit. Med. Journ.* (ii), 140.
1911	1	1	Heiduschka	*Arch. d. Pharm.*, Berl., ccxlix, 322.
—	?	?	Klausner	*Klin. Therap. Woch.*, Berl., xviii, 393.
—	1	0	Lichtenstern	*Mitt. d. Gesellsch. f. inn Med. u. Kinderh* Wien, x, 159.
—	1	1	Veress	*Budapesti, Orw. Ujság*, ix, 226, bis.
1912	?	?	Throll	*Inaug. Diss.*, Bonn.
—	?	?	Burnett and Royer	*Oklahoma Med. News Journ.*, xx, 77.
—	1	0	Earp	*Indianapolis Med. Journ.*, xv, 62.
—	2	1	Howell	*St. Bart. Hosp. Journ.*, 1912-1913, xx, 180.
—	1	0	Laehr	*Allg. Ztschr. Zeit. f. Psych.*, Berl., lxix 529-567.
—	9	6	McCrae and Colledge	*Pharm. Journ.*, Lond., 4th ser., xxxiv, 724.
—	1	1	Maclean	*Lancet* (i), 647.
—	15	2	Pollitzer	*Journ. Cutan. Dis. incl. Syph.*, N.Y., xxx, 185.
—	1	1	Rommel	*Charité Ann.*, Berl., xxxvi, 62-68.
—	2	0	Stirling	*Austral. Med. Journ.*, Melb., 1912-13, ii, 1040.
1913	3	0	Pernet	*Brit. Med. Journ* (ii), 312.
—	4	4	Vallon and Bessière	*Encéphale*, Paris, 245-261.
—	1	0	Alexander	*Brit. Med. Journ.* (ii), 20.
—	1	0	Chitty	*Lancet* (i), 917.
—	1	0	Laing	*Brit. Med. Journ.* (i), 280.
—	?	?	Rossello	*Semana méd.*, Buenos Aires, xx, pt. 2, 202-204.
—	1	1	Editorial	*Brit. Med. Journ.* (i), 566. "Veronal poisoning."
—	10	9	Willcox	*Lancet* (ii), 1178-1181. *Brit. Med. Journ.* (ii), 410.

Section of Therapeutics and Pharmacology 35

Year	Cases	Fatal	Authors	References
1914	1	0	Juarros	*Siglo méd.*, Madrid, lxi, 50.
—	1	0	Dick	*Brit. Med. Journ.* (i), 856.
—	2	1	Fraser	*Lancet* (i), 1736.
	3	3	Jansch.	Forensic Med., Alex. Kolisko (Edit.), ii.
—	24	7	Munro	*Brit. Med. Journ.* (i), 854-856.
—	1	1	Russell and Parker	*Brit. Med. Journ.* (i), 853.
—	1	0	Souper	*Brit. Med. Journ.* (i), 1015.
—	?	?	Swift	*Calif. State Journ. Med.*, San Fran., xii, 122.
—	?	?	Veronal	*N. Scuola med.*, Napoli, xxix, 8-12.
—	4	0	Glaser	*Wien. klin. Woch.*, xxvii, 1400.
1917	?	?	Fog	*Ugeskr. f. Laegar.*, Kobenk, lxxix, 370.
—	1	1	Schubiger	*Cor. Bl. f. schweiz. Aerzte*, Basel, xlvi, 1741-1750.
1918	2	2	Veronal	*Brit. Med. Journ.* (i), 182.
—	2	2	Weitz	*Med. Klin.*, Berl., xiv, 159-161.
1919	4	1	Gautier	*Rev. Méd. de la Suisse rom.*, Génève, xxxviii, 641-647.
—	1	0	Oppenheim	*Deutsche Ztschr. f. Nervenh.*, Leipz., lvii, 1-35.
—	?	?	Seldin	Inaug. Diss., Zürich, 30 p.
1920	1	0	Fleischer	*Med. Woch.*, Leipz. u. Berl., xlvi, 630.
—	1	0	Hassin and Wein	*Journ. Amer. Med. Assoc.*, Chicago, lxxv, 671.
—	1	0	Moszeik	*Med. Klin.*, Berl., xvi, 233.
—	12	2	Romer	*Deutsche med. Woch.*, Leipz. u. Berl., xlv, 1305.
—	1	0	Taub	*Journ. Amer. Med. Assoc.*, lxxiv, 459.
—	1	0	Sentis and Rimband	*Montpel. Méd.*, xlii, 52.
—	1	0	Renouf	Thèse de Paris, No. 279, 40 p.
1921	1	0	McLeod	*Med. Rec.*, N.Y., xcviii, 985.
—	1	0	Ruggles	*Journ. Nerv. and Ment. Dis.*, liv, 45-48.
—	2	0	Fremel and Herschmann	*Med. Klin.*, xvii, 716.
1922	16	5	Boenheim	*Med. Klin.*, Berl., xvii, 1263.
—	1	0	Littell	*Journ. Amer. Med. Assoc.*, lxxvii, 1333.
—	1	0	Indemans	*Nederl. Tijdschr. u. Gen.*, Haarlem, lxvi, pt. 1, 397.
—	1	0	Mantelin	*Lyon Méd.*, cxxxii, 295-297.
1923	2	1	Hertz	*Deutsche med. Woch.*, xlix, i, 416.
—	1	0	Munscheid	*Deutsche med. Woch.*, xlix, i, 690.
—	1	0	Block and Deglande	*Paris méd.*, xlv, 276.
—	1	1	Bofinger	*Deutsche med. Woch.*, Berl. u. Leipz., xlviii, 1518.
—	1	1	Cole	*Journ. Amer. Med. Assoc.*, lxxx, 373.
—	3	1	Ortner	*Wien. med. Woch.*, lxxiii, 631.
—	1	1	Kroner	*Zeit. f. ärztl. Fortbildung*, Jena, xx, 461.
—	5	0	Sands	*Journ. Amer. Med. Assoc.*, lxxxi, 1519-1521.
1924	1	0	Lespinne	*Brux. méd.*, iv, 45.
—	1	0	Tardieu and Camps	*Bull. Soc. de Thér.*, February 13, 63; also Epitome *Brit. Med. Journ.*, June 1, 88.
—	9	2	Tardieu	*Rev. de Méd.*, No. 7, xli, 393.
—	1	1	Tardieu and Medioni	*Journ. de Méd. de Paris*, xliii, 354.
—	1	0	Martindale and Westcott	"The Extra Pharmacopœia."
—	1	0	Niccolini	*Riform. med.*, Napoli, xl, 510-512.
—	1	0	Procopio	*Riv. d'ostet. e. ginec. prat.*, Palermo, xi, 241-244.
	And child			
—	3	0	Terrien	*Arch. d'opht.*, Paris, xli, 204.
—	3	2	Ipsen	*Wien. med. Woch.*, lxxiv, 2025-2028.
—	6	0	Korbsch	*Archiv für Psych.*, 1924-25, lxxii, 473-477.
—	9	4	Givkovitch-Radomir	Thèse de Paris, No. 41, 64 pp.
—	18	7	Tardieu	Thèse de Paris, No. 511, 98 pp.
—	(6 cases)	?	Kipper	*Aerztl. Sachverst. Ztg.*, Berl., xxx, 191.
1925	61	several	Leake and Ware	*Journ. Amer. Med. Assoc.*, lxxxiv, 434-36.
—	9 (+?)	1 (+?)	Caussade and Tardieu	*Bull. et Mém. Soc. méd. d.Hôp. de Paris*, 3rd ser., xlix, 295-300.
—	1	1	Rivet and Jany and Herbain	*Ibid.*, 276-279.
—	1	0	Scott	*Lancet* (i), 658.
—	5	2	Archard, Mouzon and Bloch	*Bull. Acad. de Méd.*, Paris, 3rd ser., xciii, 732-739.
—	1	0	Pasteur-Vallery-Radot and Blamontier	*Bull. et Mém. Soc. méd. d.Hôp. de Paris*, 3rd ser., xlix, 1382-1385.
—	1	1	Steindorff	*Deutsche med. Woch.*, Leipz. u. Berl., li, 1565-1567.
—	?	?	Pucher	*Bull. Buffalo Gen. Hosp.*, iii, 70.
—	1	1	Caussade, Tardieu and Lejard	*Bull. et Mém. Soc. méd. d.Hôp. de Paris*, xlix, 1485. Abstract, 1926, *Journ. Amer. Med. Assoc.*, lxxxvi, 380.

36 Young: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

Year	Cases	Fatal	Authors	References
1925	1	1	Sidney Smith	"Forensic Medicine," 373.
—	7	1	Paris Correspondent	*Journ. Amer. Med. Assoc.*, March, Discussion at meeting of Soc. méd. d. Hôp. de Paris.
1926	13	9	Redlich	*Wien. med. Woch.*, lxxvi, 139.
—	(Old cases only)		Levent	*Gaz. d. Hôp.*, Paris, xcix, 479-81.
—	1	0	Mathew	*Brit. Med. Journ.*, February 20, 323.
—	1	1	Creutzfeldt	*Ztschr. f. d. ges. Neurol. u. Psychiat.*, Berl., ci, 97-108.
—	?	?	Seymour	*U.S. Vet. Bur. Med. Bull.*, ii, 1159-1163, "Veronal psychosis."

Luminal.

Year	Cases	Fatal	Authors	References
1912	1	0	Furer	*Münch. med. Woch.*, lix, 1670.
1913	2	0	Farnell	*Journ. Amer. Med. Assoc.*, lxi, 192.
—	1	0	Pernet	*Brit. Med. Journ.* (ii), 312.
1914	1	0	Ungar	*Wien. klin. Woch.*, xxvii, 847.
1917	3	0	Strauss	*Therap. Monatsh.*, xxxi, 338.
1918	5	0	Luce and Feigl	*Therap. Monatsh.*, xxxii, 236.
1919	1	1	Hueber	*Münch. med. Woch.*, lxvi, 1090.
—	2	0	Haug	*Münch. med. Woch.*, lxvi, 1494.
—	1	1	Rosenberg	*Med. Klin.*, xv, 1150.
1920	1	0	Stein	*Therap. Halbmonat.*, xxxiv, 387.
1921	2	0	Ruggles	*Journ. Nerv. and Ment. Dis.*, liv., 45-48.
—	2	0	Fremel and Herschmann	*Med. Klin.*, xvii, 716.
1922	1	0	Phillips	*Journ Amer. Med. Assoc.*, lxxviii, 1199-1201.
1923	1	0	Hermann	*Klin. Woch.*, Berl., ii, 212.
—	1	0	Nicolai	*Klin. Woch.*, Berl., ii, 1891.
			(N.B.—Only case recorded of hippus in luminal poisoning.)	
—	9	0	von Bermuth	*Klin. Woch.*, Berl., ii, 1159.
1925	1	1	Weig	*Deutsche med. Woch.*, li, 272. Abstract *Journ. Amer. Med. Assoc.*, lxxxiv, 1159.
—	?	?	Caussade and Tardieu	*Bull. et Mém. Soc. méd. d. Hôp. de Paris*, 3 ser., xlix, 295-300.
—	0	0	Miliau	*Bull. et Mém. Soc. méd. d. Hôp. de Paris*, 3 ser., xlix, 1090-1093.
—	1	0	Caussade, Tardieu and Lacapère	*Bull. et Mém. Soc. méd. d. Hôp. de Paris*, 3 ser., xlix, 1090-1093.
—	1	0	Carlill	*Lancet* (ii), 596.
—	1	0	Weber	*Brit. Journ. Child. Dis.*, xxii, 280-285.
1926	5	1	Hamilton, Geiger and Roth	*Illinois Med. Journ.*, xlix, 344-346.
—	31	5	Schneller	*Deut. Zeit. f. d. ges. Med. Berl.*, vii, 259-277.
—	2	0	Redlich	*Wien. med. Woch.*, lxxvi, 139.

Dial.

Year	Cases	Fatal	Authors	References
1920	2	0	Müller	*Schweiz. med. Woch.*, i, 973-975.
1923	1	0	Martin and Mantelin	*Lyon Méd.*, cxxxii, 1104.
1924	1	0	Dargein and Dore	*Bull. et Mém. Soc. méd. d. Hôp. de Paris*, 3 ser., xlviii, 750.
—	1	1	Givkovitch-Radomir	Thèse de Paris, No. 41, 64 p.
1925	5	2	Achard, Mouzon and Bloch	*Bull. Acad. de Méd. Paris*, 3 ser., xciii, 732-739.
1926	1	0	Tardieu and Blondel	*Rev. de Méd. Paris*, xlii, 255-264.

Somnifene.

Year	Cases	Fatal	Authors	References
1924	1	1	Tardieu and Medioni	*Journ. de Méd. de Paris*, xliii, 354.

Medinal.

Year	Cases	Fatal	Authors	References
1920	1	0	Krause	*Berl. klin. Woch.*, lvii, 1171.
1921	1	?	Autenrieth	*Ber. d. deutsch. pharm. Gesellsch.*, xxxi, 140-146.
1922	1	0	Harris	*Lancet* (ii), 854.
1924	1	0	Korbsch	*Archiv für Psychiat.*, lxxii, 473-477.
1925	1	0	Pewny	*Wien. klin. Woch.*, xxxviii, 1360.
—	1	0	Laehr	Caussade and Tardieu, *Bull. et Mém. Soc. méd. d. Hôp. de Paris*, 3 ser., xlix, 295.
1926	1	1	Stolkind	*Lancet* (i), 391-392.
—	1	0	Mathew	*Brit. Med. Journ.*, February 20, 323.

Gardenal.

Year	Cases	Fatal	Authors	References
1926	1	0	Caussade and Tardieu and Lejard	*Journ. Amer. Med. Assoc.*, lxxxvi, 380.

Section of Therapeutics and Pharmacology 37

APPENDIX.

By Sir WILLIAM H. WILLCOX, K.C.I.E., C.B., C.M.G., M.D.

(I) *Toxicological Analyses.*

These were the analytical results in deaths from poisoning by barbituric acid compounds. The amounts found in the organs are dependent on the time which elapsed between the taking of the poison and death. In some cases most of the poison had been eliminated before death occurred. Death usually reculted from coma complicated by pneumonia.

Analyses by Mr. J. Webster, (results given in grains).

D. K., September, 1916. *Veronal.* Stomach 0·54; stomach contents 0·05; small intestine 0·35; liver 3·0; kidneys 0·8; Urine (4 oz.) 1·84.

Mrs. A., March, 1917. *Veronal.* Stomach 0·3; stomach contents 0·19; liver 3·4; kidney 1·1; blood (40 c.c.) 0·2.

J. F. L., July, 1917. *Medinal.* Stomach contents 0·3; blood (2 oz.) 0·5; liver 2·0; urine (22½ oz.) 14·0.

S. F. P., September, 1917. *Veronal.* Stomach 0·35; stomach contents 0·26; liver 3·5; kidney 0·52; urine (15 oz.) 5·2.

M. E. B., December, 1918. *Veronal.* Stomach 0·2 gr. Other organs not sent for analysis.

E. P., February, 1919. *Veronal.* Liver 0·2; kidney 0·1; stomach and contents 0·07; urine (? amount) 0·75.

Mrs. J., February, 1921. *Medinal.* Urine 10½ oz., contained 20·8 gr.

E. G. P., February, 1922. *Veronal.* Gastric contents 0·1; liver 5·6; kidney 0·6; urine (? quantity) 3·6.

W. J. T., March, 1923. *Veronal.* Stomach and contents 1·5; liver 0·9; kidneys 0·3.

L. P., March, 1923. *Veronal.* Stomach contents 0·94; stomach and portion of intestine 0·68; liver 3·5; kidneys 1·36; spleen 2·23.

Mrs. H., April, 1923. *Veronal.* Stomach 0·53; liver 2·0; intestine 1·0; kidneys 0·5.

E. S., June, 1923. *Veronal.* Stomach and contents 0·75; liver 2·0; kidneys, trace.

E. K. C., November, 1924. *Veronal.* Stomach 0·64; stomach contents 0·25; liver 2·2; kidneys 1·1; spleen 0·4; urine (2¾ oz.) 1·5.

H. W. G., February, 1925. *Luminal.* Liver, kidney, stomach and intestines together contained ¾ gr. of luminal.

A. M. S., April, 1925. *Veronal.* Liver 2·5; stomach and contents 0·8; kidneys 1·0; urine (? quantity), 7·5.

W. R. A., May, 1925. *Veronal.* Stomach-wall 5·0; stomach contents 146·0; liver 13·0; kidneys and spleen 2·0. In this case death occurred within a few hours of taking the poison. A very large dose was taken as shown by the enormous quantity found in the stomach contents at the time of death.

Analysis by Sir William H. Willcox.

E. H. T., September, 1912. *Veronal.* Liver 2·0; kidneys 0·56; intestines 2·87; brain 1·28; blood (9 oz.) 0·87 = Total 7·58 gr. Death occurred thirty-two hours after taking the fatal dose of veronal.

(II) *Cases of Acute Poisoning by Barbituric Acid Compounds.*

(Seen by Sir William H. Willcox).

(1) G. S., male, aged 37, July 5, 1912. Large dose of veronal taken July 1, 1912, after which he had to be awakened. Probably further doses taken on July 4, 1912. Patient found comatose on July 5, 1912. On July 6, 1912, deep coma, temperature 105° F. Signs of broncho-pneumonia both lungs (dullness, tubular breathing, moist sounds). Knee-jerks just obtained. Plantar reflexes flexor. Temperature between 103° F. and 105·8° F. till death July 8, 1912. This patient had been addicted to the use of veronal.

(2) W. J. S., male, aged 62, August 28, 1921. Patient took 53 gr. of veronal on August 27, 1912, and became comatose a few hours afterwards. On examination (August 28, 1912),

temperature 102·8° F. Signs of broncho-pneumonia in left upper lobe (dullness, tubular breathing and crepitations). Some moist sounds also to right base. On August 29, 1912, temperature fell to normal and patient regained consciousness. Recovery occurred. The patient had been addicted to the use of veronal and other drugs for insomnia. This is the only case I have seen in which recovery occurred after well-marked signs of pneumonia have developed as a complication of veronal poisoning.

(3) Mrs. H., June 2, 1915. A veronal addict. 125 gr. of veronal taken 9 a.m. June 2, 1915. Stomach washed out at 10.3 a.m. Treatment: caffeine sod. salicylate gr. ii, eight-hourly, hot coffee by gastric tube, saline colon washes. Patient comatose. Knee-jerks present left side, absent right side. Plantar reflexes flexor. Patient became conscious after twenty-four hours and made a good recovery. (The prompt gastric lavage undoubtedly saved the life of this patient.)

(4) Mrs. J., aged 52, February 21, 1921. Patient had been taking medinal gr. v nightly over a long period. On February 20, 1921, took 200 gr. of medinal. Coma developed within an hour and continued until death. On February 21, 1921, definite signs of broncho-pneumonia were present. Death occurred February 22, 1921.

(5) F. W. B., male, aged 30. November 3, 1921. A large amount of veronal (probably about 150 gr.) taken at bedtime on November 6. Patient found comatose November 7. On November 8, temperature 102·6° F., deep coma, signs of broncho-pneumonia, death 8.30 p.m. November 8, 1921.

(6) Mrs. H., aged 40, October 2, 1922. In habit of taking veronal. On October 1, 1922, 10 gr. veronal taken 10 p.m. On October 2, 1922, 50 gr. taken in early morning. At 10 a.m. patient seen by Sir Bruce Bruce-Porter, who found great drowsiness, diplopia, indistinct speech and ataxy present. Stomach washed out. Patient became comatose at 11 a.m. and remained so till 9 p.m. Knee-jerks just present on each side. Plantar reflex extensor left side, doubtful response right side. At 3 p.m. lumbar puncture and cistern puncture by Sir James Purves-Stewart. Patient made a good recovery. The removal of cerebro-spinal fluid appeared to accelerate the return of consciousness. Analysis of urine by Mr. John Webster, F.I.C., taken on October 3, 1922, showed veronal $1\frac{1}{4}$ gr. in $7\frac{3}{4}$ fluid ounces. Cerebro-spinal fluid (cistern puncture) $\frac{1}{2}$ oz. had 0·043 gr. veronal. Cerebro-spinal fluid (lumbar puncture) $\frac{1}{2}$ oz. had 0·025 gr. veronal.

(7) L. G., male, aged 60. September 16, 1923. Had been addicted to veronal and dial habit for two years. Had been told these drugs were harmless. Had noticed his gait was unsteady the last six weeks. Had been very depressed lately, and says he took the drug in a big dose because of depression. On September 14, 1923, 1 a.m., took 60 gr. of veronal, found comatose several hours afterwards, recovered consciousness on September 15, 1923, and made a good recovery.

(8) Mrs. E. K. C., aged 41. November 25, 1924. Had been in habit of taking veronal for insomnia. On November 24, 1924, 9.30 p.m. locked bedroom door. On November 25, 1924, 12.30 p.m. patient found in deep coma. At 8 p.m. deep coma, temperature 102·6° F., marked signs of broncho-pneumonia. Death occurred November 26, 1924, 11.15 a.m. Probably about 100 gr. of veronal taken. Analysis *vide* list in Appendix, E. K. C.

(9) Mrs. A., aged 35. March 19, 1925. In habit of taking medinal at night. On March 19, 1925, at 4 p.m. took 80 gr. medinal, at 4.30 p.m. found comatose. Stomach washed out 6 p.m. Patient was comatose for several hours and then made a good recovery. The washing out of the stomach no doubt saved the life of this patient.

(10) W. H. G. S., male, aged 50. February 22, 1927. Patient had been addicted to taking veronal at night for years. On February 20, 1927, about 11.30 p.m. took a large dose of veronal. On February 21, 1927, found in deep coma. On February 22, 1927, 2 p.m. deep coma, temperature 100° F., signs of broncho-pneumonia in both lungs. Knee-jerks absent. Plantar reflexes flexor. Death on February 23, 1927.

(11) H. C., male, aged 50. Admitted St. Mary's Hospital, April 9, 1920. On April 4, 1920, found in heavy sleep at hotel, and was roused with difficulty. Got up and was about till April 8. On April 8 told maid he would take "a large dose." On April 9, 1920, found comatose in hotel, admitted to hospital in state of deep coma, with Cheyne-Stokes respiration. Knee-jerks and plantar reflexes absent. Marked signs of broncho-pneumonia. Death April 10, 1920. Veronal found on analysis.

(12) C. B. M., female, found comatose at hotel on May 31, 1921, at 5.30 p.m. In bedroom from 7 p.m., May 30, 1921. On June 1, 1921, deep coma, knee-jerks and planta-

reflexes absent. Marked signs of broncho-pneumonia. A blotchy red rash on skin ? erythema on folds of elbows. On June 2, 1921, temperature 105° F., deep coma. Feeble pulse, death 6 p.m. Veronal found in bedroom. Post-mortem, marked signs of broncho-pneumonia. Cloudy swelling of liver, kidneys, and myocardium. Veronal found on analysis.

(III) *Cases of Chronic Poisoning by Barbituric Acid Compounds.*

(1) P., female, married, aged 35. Had been taking veronal for some months and was definitely addicted to drug, which was withheld with difficulty. Mental depression. Marked ataxic gait, visual hallucinations were present. On discontinuance of the drug the symptoms completely cleared up after about three weeks.

(2) W., female, married, aged 70. June 25, 1924, drowsy for six weeks, speech indistinct, diplopia, slight ptosis on right side, gait ataxic with tendency to fall to left side. An organic brain lesion was suspected in this case and most careful investigations made. It was found that the patient had been taking veronal 10 gr. nightly for a long period unknown to her doctor. On discontinuance of the drug the nervous symptoms completely cleared up.

(3) G., female, aged 50. Acute sciatica on left side for ten days. Treated with dial, 1 tablet three times daily for three days. On examination patient was drowsy, speech indistinct, tongue furred and could not be protruded. Could not move left arm. Retention of urine for twenty-four hours. These symptoms were undoubtedly due to dial poisoning, and completely cleared up in forty-eight hours after purgation, hot coffee and colon washes.

(4) Case X, married, aged 55. On examination patient was drowsy, gait ataxic, diplopia, defective articulation and on one occasion showed extensor plantar reflex on both sides. The urine contained veronal, 2 gr. in 3¼ oz. The cerebro-spinal fluid contained $\frac{1}{10}$ gr. of veronal in 4 c.c. The symptoms completely cleared up after six days. The case was undoubtedly one of chronic veronal poisoning and veronal compounds had been taken over a period of some months. Sir James Purves-Stewart saw this patient with me and concurred in the diagnosis.

(5) A., male, aged 34. Previous history of alcoholism. Had taken no alcohol for seven weeks, but during this period, while under medical observation, had taken three or four tablets of allonal (2½ gr. in each) every night for insomnia. On examination speech thick and indistinct. Has had diplopia in mornings for last few days, gait ataxic. This patient was also seen by Sir James Purves-Stewart who concurred in the diagnosis of allonal poisoning. The symptoms disappeared after the drug had been discontinued.

Discussion.—Dr. W. E. DIXON, F.R.S., said that it was difficult to explain why persons suffering from drug poisoning should succumb to pneumonia. What was the mechanism of this? Did the leucocytes become paralysed? Veronal did not go the lungs, and yet a lung lesion was produced. The selective action of the drugs in causing paralysis in one limb rather than in the other was noteworthy.

Dr. F. PARKES WEBER referred to an unpublished case of hæmatoporphyrinuria undoubtedly due to *veronal*. In September, 1923, he saw a woman, aged 39½, recovering from a very painful organic illness (meningococcal meningitis), who for more than two months had been given five grains of veronal almost every night as a hypnotic. Her urine for a considerable time had been either deep red or red-brown in colour, free from hæmoglobin and albumin. The colour was due to the presence of hæmatoporphyrin (kindly confirmed by Dr. Mackenzie Wallis), and disappeared gradually in the course of two weeks after the discontinuance of the veronal. For a time the patient also suffered from paresis of the lower extremities (right knee-jerk absent), with pains in the calf-muscles, possibly due to so-called hæmatoporphyrinuric polyneuritis.

Dr. Weber also referred to the case of a choreic girl, aged 13 years, who after ordinary doses of *luminal* developed a florid morbilliform eruption (slightly purpuric in parts) accompanied by bullous formation. The contents of the bullæ were deep ochre-yellow from the presence of bilirubin and gave a strongly positive biphasic Hijmans van den Bergh reaction. Soon afterwards the jaundice became more obvious and the sclerotics had a definite yellow tinge. Both the eruption and the jaundice gradually disappeared although the jaundice became at first more marked after the luminal had been discontinued.[1]

[1] F. Parkes Weber, "A Case of Morbilliform and Bullous Eruption with Jaundice, from Luminal, in a Choreic Child," *Brit. Journ. Child. Dis.*, London, 1925, xxii, 280.

40 Willcox: *Hypnotic Drugs of Barbituric Acid & Sulphonal Groups*

The pneumonia from veronal poisoning, mentioned by Sir William Willcox, might (apart from aspiration-pneumonia—"Schluckpneumonie") be due simply to exposure during deep sleep or coma. He (Dr. Weber) thought that exposure to cold during deep sleep of any kind was occasionally followed by pneumonia—at any rate in some individuals. The extensor type of plantar reflex noted in certain cases of acute veronal poisoning mentioned by Sir William Willcox, might also be due merely to the deep sleep or coma. Probably any kind of very deep sleep or coma might in some individuals give rise to a positive Babinski phenomenon.

Dr. P. HAMILL said that there was great difficulty in diagnosing veronal poisoning. Had Sir William Willcox actually observed similar toxic phenomena occurring with the new drugs of the barbituric acid series, because in a certain number of cases drugs of this type did relieve pain in a remarkable manner?

Professor J. A. GUNN said that veronal lowered the bactericidal power of the blood.

Dr. E. STOLKIND said that some authors regarded medinal or sodium salt of veronal as a harmless drug, even for children. Though there were on record hundreds of cases of veronal poisoning, there were comparatively few due to medinal. On this account the case he was about to report of chronic and acute fatal medinal poisoning assumed great interest. The patient, a literary man and good public speaker, became an addict to medinal and was unable to give it up during the last eight years of his life. It was most instructive to watch the ill-effects of this drug habit on his physical and mental condition. Gradually he became thinner and weaker; there was oliguria with passing of urine once or twice in twenty-four hours. His thinking, speaking, and writing capacity, as well as his movements, grew slower and slower. There were other symptoms as well—ataxy, tremor, etc., and sometimes incoherence and delusions. By mistake he took an overdose of medinal and died thirty-nine hours later. The symptoms were the same as in cases of acute fatal veronal poisoning. Congestion and œdema of the lungs set in only during the last hours of life. His (the speaker's) conclusions were that medinal had the same cumulative toxic effects as those of veronal; and that the chronic medinal habit produced the same after-effects on the physical and especially on the mental condition as veronal, the ill-effects of which might be compared with those of morphinism.

For the last seven years he (Dr. Stolkind) had used luminal for a great number of out-patients, adults and children, suffering from different forms of epilepsy. At first he gave only small doses—1½ gr., but later he increased this to 2½ gr. twice daily. In many cases of "idiopathic" epilepsy the results were quite beneficial. Often there had been no recurrence of the fits for years. Some patients who had not benefited from years of bromide treatment, became quite different persons after taking luminal. In spite of giving luminal for years, he (Dr. Stolkind) had had only three cases (one with icterus) in which there was eruption. No other complications were observed. In cases of epileptic fits, e.g., due to dyspituitarism, luminal had frequently had no effect.

The law allowed everyone to buy quite freely, at the chemist, every hypnotic drug mentioned by Sir William Willcox. It was only necessary to sign one's name in the book to procure such drugs, no doctor's prescription being required.

Dr. DOROTHY HARE said that in giving luminal in epilepsy she had never exceeded 2 gr., but recently, in one case, after a long spell of treatment, she noticed a decided lethargy which she, at first, thought to be a mild attack of encephalitis lethargica; she now, however, wondered whether it might have been mild luminal poisoning.

Sir WILLIAM WILLCOX (in reply) said that luminal was very toxic, and would cause the same changes as had been described. Luminal and the members of the barbituric acid group were not cumulative in the material sense, for they were rapidly excreted by the kidney, but the effects on the nervous system were cumulative. Why pneumonia occurred in veronal poisoning he did not know. He had always put it down to the abolition of the reflexes in the upper respiratory tract, which led to the passage of foreign substances into the lung. As for the new drugs of this group issued under different names, he believed that they did cause toxic symptoms; he had had several letters to that effect, and had himself seen cases.

Section of Tropical Diseases and Parasitology.

President—Dr. R. T. Leiper, F.R.S.

Some Features of Crown Gall in Plants in Reference to Comparisons with Cancer.

By Wilfrid Robinson, D.Sc.

(Professor of Botany, University College of Wales, Aberystwyth.)

From time to time comparisons have been made between tumour-like growths in plants, and tumours, benign or malignant, in animals and man. Such comparisons, when carefully analysed, have usually proved to be more superficial than real, if only because of the profound differences in the cellular construction and manner of growth in animals and plants. In recent years, however, the bacterial disease of plants known as *crown gall* has been very persistently compared with malignant tumours in man. The disease has been the subject of much work and of many papers by Dr. Erwin Smith in America, who first isolated the causal organism *Bacterium tumefaciens* and experimentally produced the disease by the re-inoculation of plants from pure cultures.

Dr. Smith soon formed the opinion that the growths characteristic of this disease are comparable to malignant tumours. In his earlier work the evidence for the far-reaching comparisons with cancer was derived from artificially-produced growths which, in addition to having primary swellings near the region of inoculation, showed others, described as secondary tumours, which were connected with the former by so-called tumour-strands. The parallel was thus drawn with the origin of secondary tumours in malignant diseases in animals by metastasis from the primary tumour.

Though able to isolate bacteria from the tumours, Smith failed to locate the former in the diseased tissues by direct microscopic observation. More recently Blumenthal and others working with him in Germany have isolated from the margins of human tumours a bacterium which gives cultural reactions almost identical with those of *Bacterium tumefaciens* (E. F. Smith). Further, at least, one of the strains isolated from human tumours has proved capable of producing, on sunflowers, galls identical with those of crown gall. In addition Blumenthal claims to have produced tumours in healthy animals by inoculation with the same organism.

I shall re-describe briefly some studies carried out by Mr. Walkden and myself at Manchester, on crown gall, using a strain of *Bacterium tumefaciens* isolated by Mr. Walkden from diseased plants of chrysanthemum found in gardens. From our results I shall endeavour to give reasons why Smith's comparisons cannot be regarded as valid, also, in my opinion, why the work of Professor Blumenthal and his colleagues cannot be accepted as proving that an organism similar to *Bacterium tumefaciens* is responsible for malignant diseases in animals and man. Finally, I hope to offer some suggestions as to a possible fundamental resemblance between crown gall and animal diseases in which cell-proliferation is the dominating feature. In doing this I shall have to refer to the recent illuminating researches of Warburg, in Berlin, in support of the view that, while the comparisons of Smith, Blumenthal, and others, cannot fruitfully be carried further, the study of the intimate cellular physiology of proliferating growths in both plants and animals gives promise of an ultimate solution of many of the problems of malignant disease.

54 Robinson: *Crown Gall in Plants in Reference to Cancer*

Our work on crown gall will now be dealt with, and it must be emphasized that the so-called secondary tumours described by Smith do not occur naturally on plants but result from artificial inoculations. The naturally occurring galls on the common garden marguerite (*Chrysanthemum frutescens*) are globular swellings with a rough surface bounded by superficial layers of dead cells. Such galls have the bacteria present in abundance on the surface. In our work the method adopted was the inoculation of the cut ends of shoots with pure cultures of the bacterium, since this organism can only produce galls after wounding.

The bacteria enter the intercellular spaces of the tissues as well as the open ends of the vessels passing down the shoot, for a smaller or greater distance. Adjoining the lines of invading bacteria, cells and tissues, normally inactive, are stimulated to repeated division—subsequently either continuing their growth or differentiating into irregularly arranged woody tissue. Control shoots cut similarly but not inoculated gave no such results.

It was thus demonstrated that the presence of the bacteria on the exterior of galls and in the air spaces between the cells (they have never been recognized within the living cells) influence these cells to active proliferation. The possible nature of the influence exerted will be referred to below. In such rough-surfaced galls, which correspond to the primary tumours described by Smith, the later development of the gall is partly due to the active presence and multiplication of *Bacterium tumefaciens* on the rough external surface of the gall. It was found possible to repeat most of the work of Smith on the so-called secondary tumours, but the results obtained show that the facts will not bear Smith's interpretation.

In the marguerite the so-called secondary tumours arise when the inoculations are made by needle-pricks into the vicinity of the growing points or apical meristems of actively growing shoots. A number of leaf-rudiments as well as the meristematic tissues of the stem are affected by the needle-prick, and by the bacteria carried in by it; and as the growth of the plant proceeds the injured parts which develop galls are widely separated from one another by the growth of the shoot. Many such galls have a rough exterior, but others are smooth, and these latter correspond to Smith's secondary tumours. They are not, however, due to the intrusion of tumour-strands as he suggested, but have been shown in our work to be due to the presence of bacteria in the air spaces between the tissues. These bacteria have been proved either to be carried by the growth extension of the tissues to a distance from the original point of entry, or to migrate actively in the intercellular spaces. Although regions infected by the migrating strands of bacteria superficially resemble invasive tumour strands, it has been demonstrated that cells are merely altered *in situ*, there being no migration of the cells of the diseased plant. The bacteria, on the other hand, move within the spaces as a zooglœal thread.

This migration of the bacteria can be observed particularly easily in tobacco plants, if the young flowering shoots are cut across and the wounded surface then inoculated with *Bacterium tumefaciens*. A rough gall arises at the cut surface and a string of smooth galls, with healthy tissues between, occurs for some distance below the cut surface. As cell-proliferation proceeds around the loci formed by the invasive zooglœal threads some of the pre-existing tissues may be somewhat displaced by inequalities of growth, but there is never any true metastasis.

Other features of crown gall, such as the production of apparent teratomata and the apparent transformation of the structure of one organ into another as a secondary effect of the bacterial activity, have been shown to be equally invalid as the bases for any far-reaching or real comparison of crown gall with malignant tumours.

Experimental work by Riker in America, and by Kuster in Germany, has independently led these investigators to conclusions similar to our own; others, in

Germany and France, approaching the subject from the point of view of malignant disease, have come to the extraordinary conclusion that *Bacterium tumefaciens* or organisms very similar to it occur in tumours in man, and that when isolated these organisms prove capable of producing not only galls on plants but tumours on animals. I find it difficult to accept Blumenthal's results as conclusive, for several reasons. In no case in which tumours occurred in animals after inoculation with the strains isolated from human tumours did Blumenthal succeed in re-isolating the inoculated bacteria from the tumour in the diseased animal. Until this has been done the causal connexion between the organism inoculated and the growth produced cannot be regarded as established. Blumenthal himself explained the discrepancy in the evidence by suggesting that an ultramicroscopic virus is associated with the bacterium in those cases in which inoculated animals develop tumours. In this way Blumenthal possibly foreshadowed the ultramicroscopic theory of Gye and Barnard.

Feggins and Funk in France have claimed more recently that they have isolated from a uterine tumour a bacterium, which, on inoculation into pelargonium plants, proved in one case capable of causing the development of galls as large as a hazel nut.

The fact that it should be possible to isolate organisms similar to *Bacterium tumefaciens* from malignant tumours is in itself not surprising in view of the well-known presence of a variety of bacteria on the necrosing margins of tumours. Such bacteria have of course no causal connexion with the tumours. It may therefore not be without significance that Blumenthal's isolations were made from the secretions of the marginal regions of tumours subjected to a special treatment with sun's rays, and that several other species of bacteria, in addition to the strains similar to *Bacterium tumefaciens*, were isolated.

There thus appears to me to be little, if any, justification either for the comparisons instituted by Smith, or for the conclusions arrived at by Blumenthal regarding a possible connexion between the causal agent in crown gall and malignant tumours. There are, however, certain features which the plant and animal diseases share in common. For example, in both cases, cells and tissues are stimulated to active atypical proliferation, and I have suggested elsewhere that a real insight into the nature of the changes taking place under the influence of *Bacterium tumefaciens* might throw light on the changes occurring as the result of unknown causes in the cells of animal tumours. The recent work by Warburg, mentioned earlier, bears directly on this aspect of the subject.

Rejecting the theories of Gye as well as those of Smith and Blumenthal on the ground of the lack of clinical evidence of the infectious nature of malignant tumours, Warburg holds that the problem is essentially one in cellular physiology. He has studied, by exact experimental methods, the energy-liberating reactions in rat sarcomata and carcinomata, and in fowl sarcoma, measuring the fermentative changes in the tumour tissue. He finds that this fermentative activity resulting in lactic-acid production is a property of all non-necrosing tumour tissue whether from animals or from man, and that such tissue shows this property irrespective of the presence or absence of oxygen. In the absence of oxygen all body cells show fermentation, but Warburg has proved that only embryonic tissues, e.g., of the hen's egg, show a fermentative activity, in the absence of oxygen, at all comparable in magnitude to that of tumour tissue. In the presence of oxygen this is masked in the healthy embryonic tissues by active respiration. Thus Warburg concludes that in tumour tissues the oxygen respiration of growing cells is electively injured, while the fermentative activity still proceeds. This can be illustrated experimentally by placing an embryo for some time in nitrogen and then once more in oxygen. The respiration is injured, but the fermentation is unaffected, i.e., the metabolism characteristic of tumour tissue persists. Poisons and

other injurious substances act similarly and, *in vitro*, tumour cells occasionally arise. Usually, however, in experiments, as in the body, the effect of injuring respiration is to cause the death of the cell.

In many cases in plants it is well known that lack of oxygen for a time may result in cell-proliferation, and it may be significant in crown gall that *Bacterium tumefaciens* is an organism which is very strongly aerobic. Thus it is possible that in the intercellular spaces it modifies the oxygen relations of the cells so that the internal changes resulting in proliferation are set up. Whether the tissues in crown galls show a similar type of metabolism to that which Warburg has found in animal tumours will be seen from further work. It certainly appears possible that in their cellular physiology the plant galls may have at least some points in common with the animal tumours.

(*May* 4, 1927.

The Development of *Schistosoma mansoni*.

By Dr. P. H. J. LAMPE, Surgeon to the Dutch-Indian Army.

(*Communicated from the Pathological-anatomical Laboratory of the Military Hospital of Paramaribo, Dutch Guiana.*)

DURING an investigation into the development of *Schistosoma mansoni* at the beginning of this year, undertaken more out of interest in the cause of a disease endemic here than with the intention of discovering anything new in connexion with it, a few details were observed, some not yet mentioned in the literature of the subject and some not entirely in keeping with facts therein stated.

Therefore, notwithstanding the excellent work of Leiper on the development in the intermediate host, and that of Yamagiwa, Lütz, Faust, and others, on the development in the final host, I venture to mention briefly certain observations of my own in Surinam on the development of this highly interesting trematode.

(I) THE DEVELOPMENT IN THE INTERMEDIATE HOST.

The full-grown parasites of this species of Schistosoma live in the hepatic portal vein and its branches. The parasites—paired—swimming against the blood-current, enter the narrower veins, where the thinner female, quitting the male, deposits its ova.

The ova, distinguished by a lateral spine, measure about 165 microns, and the spine about 26 microns. In order to continue the species, a number of the ova penetrate the intestinal wall and reach the intestinal canal.

The part played by the lateral spine is not quite understood. Possibly the ovum is forced back against the intestinal wall by the blood-current and remains sticking to it by means of this lateral spine. With the fæces the ova are ejected.

If the ovum be placed in a hypotonic medium (e.g., water, or very diluted eosin-solution, added to the fæces) the embryo, if alive, visible through the egg-shell, starts moving, while, after an interval—depending on light, temperature (optimum 35°) and unknown influences—the shell ruptures laterally somewhat behind one of the poles.

The miracidium slowly creeps out, and, escaping from the shell, swims away. I have never observed in the case of *Schistosoma mansoni* the dilatation of the egg-shell as described by Brumpt for *Schistosoma hæmatobium*—"L'œuf se gonfle."

The duration of the life of the free swimming miracidia—estimated at two to three days—is shown in Surinam (laboratory temperature 33° max.) to be forty hours at the longest: according to Faust, in China, three days (*Schistosoma japonicum*); according to Christopherson, in the Sudan, at the most nine hours

(*Schistosoma hæmatobium*). The morphological structure is clearly discerned by staining with eosin, especially in dying forms, as a mobile protoplasmic mass, ever changing in shape, clothed with cilia, the greatest measurements being between 110 and 240 microns. At one pole is found a headlobe or papilla surrounded by a ring of cilia. The germ-cells, salivary glands, cerebral ganglion commissures, the blind, pouch-like intestine and the excretory apparatus are clearly seen.

The ova being deposited with the fæces in water frequented by certain species of snail, further development takes place within these molluscs.

According to Leiper, who visited Surinam in 1921, *Planorbis olivaceus* is found in this colony exclusively. He found 5 per cent. of these snails infested with *Schistosoma mansoni*. After a systematic examination of some thousands of snails from infected areas around Paramaribo, I found that not only *Planorbis olivaceus*, but also *Planorbis guadeloupensis* is found naturally infected in the locality.

The highest percentage of infested *Planorbis olivaceus* was 18 per cent. for the month of September, that of infested *Planorbis guadeloupensis* 15 per cent. for the month of July (1925).

Observations published hitherto on the penetration of the snail by the miracidium are more or less in agreement. The miracidia are said to pierce the soft parts of head and foot (Miyairi and Suzuki, 1913, Franco and Mello, 1921, Faust, 1924), of the tentacles (Lütz, Brumpt and others, 1917) or of the mantle-cavity (Manson-Bahr and Fairley, 1920).

My observations do not entirely agree with these authors' views. Under the binocular microscope the attraction of the miracidia can be clearly observed. Only a few adhere to the external soft parts of the snails; the bulk disappear into the buccal and the mantle cavities. I have never observed the mutilation of the tentacles, described and illustrated by Lütz and Brumpt, which would be caused by a mass invasion of these organs. I seldom found miracidia in the tissue, in serial microscopic sections, although they were prepared immediately after the infection was definitely observed under the microscope.

Either the miracidia have certainly penetrated, and have not been found, even when many serial sections have been made, or they have been observed in the buccal or the intestinal canal. Therefore I think it justifiable to assume that infection of the snails through the mouth is not only possible, but may be considered most frequent.

The infection-method of other trematodes, the miracidia of which do not spontaneously escape from the egg-shell, but are liberated in the stomach of the intermediate host (*Clonorchis sinensis, Distomum macrostomum, Dicrocœlium lanceolatum, Opisthorchis felineus*) is analogous and fully justifies this statement.

Further development in the snail is easily traced. The unchanged miracidium that has penetrated, as a rule—even in sections made immediately after the infection—is difficult to find; the other stages may be clearly seen in the microscope sections. After a few days a sporocyst in its first stage of development can be detected as a thick-walled pouch in or close to the intestinal wall. As this primary cyst steadily grows it changes into a thin-walled tube constricted in places; daughter-sporocysts, which develop from local thickenings of the mother-sporocyst wall ("germ-cells"), arise; they are isolated from the wall, and eventually fill the mother-sporocyst. In these daughter-sporocysts, regenerating by transverse fission and furrowing, the so-called cercariæ develop again out of local thickenings of the wall (buds). The danger to man lies with these cercariæ.

The moment of the rupture of the primary cyst varies in different individuals. In the third, or at the beginning of the fourth week, the liberated daughter-sporocysts are found between the hepatic islands, in the form of thin-walled pouches, filled with tail-less cercariæ. In these wandering daughter-sporocysts reproduction is still going

on by transverse fission and furrowing. The hepatic tissue degenerates in consequence of pressure-atrophy. The thin wall of the daughter-sporocysts expanding approaches the mantle-cavity of the snail, and at the spot of least resistance when the cyst is entirely filled with cercariæ it ruptures. The cercariæ escape in bunches into the mantle-cavity. From here they swim away into the surrounding water.

The number of cercariæ produced by an infested snail during its life-time of two or three months is so fabulously large that it can hardly be estimated. For instance, from a snail infected in the laboratory 600 cercariæ per hour escaped on the thirty-seventh day after the infection (7 a.m. to 5 p.m.), 92 per hour on the fifty-sixth day, 190 per hour on the fifty-seventh day, and 44 per hour on the seventy-eighth day. During night-time cercariæ do not escape.

From many controlling observations on an artificially infected snail, from which cercariæ continued to escape during sixty-five days, I was able to estimate the number approximately at 172,000 (2,646 daily). Liberation is enhanced by light and heat, under the influence of which the snail moves about with activity.

The lifetime of these cercariæ in pure water at laboratory temperature is more than twenty-four hours. In a suspension of red-blood cells in physiological NaCl solution, at body temperature, it is not more than six hours.

Excepting sections in series the different stages of development of infected snails are most clearly demonstrated by a binocular microscope. The snails are macerated in a mixture of glycerine and chloral hydrate 3/7 (Vonk) in which they remain some days. The isolation of sporocysts and daughter-sporocysts, otherwise not possible, is easily effected by this method.

The duration of this parthenogenetic development[1] was found in our laboratory to be never more than thirty-four days and never less than thirty-three days.

Experiments were carried out to discover what influence light and temperature have on the period of development. To that end a large number of snails infected at the same time (*Planorbis olivaceus* and *Planorbis guadeloupensis*) were kept in different groups under different conditions. The result was that the period of the development proved practically independent of the influence of light and temperature. The mortality of the infected snails is greater at high temperature and less with snails kept in the dark, than under ordinary conditions.

Lütz, in Brazil, saw the first cercariæ escaping after 30 days (*Planorbis guadeloupensis* and *Schistosoma mansoni*). Cawston, in South Africa, mentioned that the first cercariæ never escape before the thirty-fifth day after artificial infection (*Physopsis* and *Schistosoma hæmatobium*). Faust and Meleney, in China, found the duration of the development in the snail to be about seven weeks (*Onchomelania hupensis*, *Schistosoma japonicum*).

(II) The Development in the Final Host.

These experiments were carried out with two different species of cercariæ of the order of the Schistosomidæ, viz., with the larvæ of *Schistosoma mansoni* and with a furcocercous cercaria of different size, without pharynx and eye-spots, derived from *Planorbis guadeloupensis*.

The measurements of the living cercariæ were as follows:—

	Cercaria of *Schistosoma mansoni*	Cercaria of unknown species
Body	75 to 225 microns ...	75 to 150 microns
Unforked tail ...	150 to 225 „ ...	125 to 150 „
Tail fork	90 „ ...	180 „

Fixed in 10 per cent. formalin, these measurements became:—

Of cercaria *Schistosoma mansoni* 115 microns, 190 microns, 38 microns
Of cercaria spp. ... ... 120 „ 125 „ 150 „

[1] The germ cells being considered as parthenogenetically developing ova.

These measurements of *Schistosoma mansoni* do not agree with the numerous and variable statements published.

Guinea-pigs, rabbits, cats, mice, ducks, chickens, etc., were used for experiments.

Method of Infection.

(1) Submersion of the partly shaved or plucked animal in highly infected water. (2) Pouring of water, containing cercariæ, into the mouth. (3) The same in the conjunctiva. (4) The same through the anus into the rectum. (5) Subcutaneous injection with water containing cercariæ.

Precautionary measures were taken :—

(1) To avoid the skin being punctured during shaving or plucking, which might facilitate penetration. (2) To avoid cercariæ coming into contact with the skin during the artificial infection by way of the mucous membranes.

At intervals after the infection the mammals were killed, and the organs (*porte d'entrée*, heart, lungs, diaphragm, liver, spleen, kidneys, brains, intestine and mesentery) microscopically examined.

(1) *Infection through the Skin.*

Infection through the skin proved successful. At first it was difficult to find the cercariæ which had penetrated in the skin. Even by violently exaggerating the conditions appertaining in nature, e.g., submerging the ear previously macerated, in strongly infected water for many hours, cercariæ which had penetrated were looked for in vain in a large number of serial sections. The water used for the infection showed, however, that infection had taken place, as many immobile tails minus bodies were found in it.

These experiments were frequently carried out and the results were always uniform. I therefore assumed that the relatively few cercariæ that do penetrate remain in the skin and the subcutaneous tissue only for a short time. The results of repeated investigations confirm this assumption. If the duration of the infection was shortened to fifteen to thirty minutes it proved easier to find the cercariæ in the skin. The results were somewhat different when the skin was incised ; the number of cercariæ left behind was smaller, the number of the tails abstracted larger, and the cercariæ were easier to find in the skin.

The best results were obtained when experimenting with new-born mice, which could be bodily submerged in the infected water and microscopically examined. In these experiments nearly all cercariæ entered the body of the subjects of experiment, the great quantitative difference being undoubtedly due to the tenderness of the skin of the new-born animals.

These latter experiments also showed that the cercariæ do not make use of the hair-follicles or pores of the skin. Sections show that the skin—perhaps after previous lysis (by the action of the salivary glands of the cercariæ)—is severed and lifted up.

In view of these experiments I think that, under natural conditions, infection through the intact skin of the body or extremities of adult men or animals does not easily take place. This is not to be considered as contrary to the generally accepted opinion that the skin is the most frequent *porte d'entrée*. The tender skin of the præputium, the soft skin between the toes, and also the frequently present lesions or wounds on the extremities, offer sufficient chances for the infection of peasants and labourers working in swamps and morasses, and also for infection of careless bathers.

Moreover the question of the difficulty of the penetration of the experimental animals by the cercariæ should be duly considered, viz., as to whether this difficulty might not arise from the unsuitability of the animals submitted to infection.

With regard to these experiments with mammalia, it may be stated that newborn mice were easily infected, the mucous membrane of large animals succeeded well, and cercariæ injected subcutaneously always reached complete development.

(2) *Infection through the Mucous Membranes.*

This mode of infection always produced good results. Considering the fact that the cercariæ need not enter through hair-follicles or pores, this was to be expected.

(3) *Subcutaneous Infection.*

This required great care. When too many cercariæ are injected, the animal dies, generally after a few days, with diffuse infiltration of both lungs (see later on). If this stage be passed, death ensues after some weeks in consequence of a blocking up of the small ramifications of the hepatic portal vein by young parasites.

(4) *Experiments with Birds.*

As might be expected, these cercariæ of mammalian trematodes never attained a full development when birds were used for experiment. Birds were infected in order to discover whether these cercariæ had any tendency to penetrate extraneous animals. With birds the infection through the intact skin proved successful, and infection through the wounded skin and mucous membranes (*per anum*) readily produced definite results.

It is not impossible that this infection of an extraneous animal plays an important part in the epidemiology of this disease (*diversion of parasites*), the more so because the cercariæ that penetrate never arrive at sexual maturity or undergo further reproduction.

(5) *The Route by which the Penetrating Cercariæ reach the Vena Porta.*

This was always the same in the case of both species of Schistosomidæ and was used with all experimental animals.

The cercariæ, in whatever way they may be brought into contact with the experimental animal, always find their way into the veins, after having wandered for some time within the connective tissue of the skin or mucous membrane. They then immediately feed on the blood; a few remaining behind in the tissue die. The majority reach the veins and so the right heart. Six hours after infection cercariæ may be found in coagulated blood in the heart. Serial sections of the heart show, however, that the cardiac muscle or the septum is never pierced.

Cercariæ, once in the blood-circulation, find their way into the lungs. The lung is therefore a good place in which to find cercariæ. In the lungs, they leave the thinner branches of the pulmonary artery, and then—filled with blood—penetrate the stroma between the alveoli. At first they were found diffusely spread; later on they accumulated in the bases of the lung.

Microscopic sections of apices and bases of lung show a marked difference two or three days after the infection. Most of the cercariæ are then congregated in the bases of the lungs. This, it seems, is the critical period of the development of this parasite. Except for a steady growth and further aggregation in the pulmonary bases, further migration into other organs does not occur in the majority of cases. They wander about within the lungs and die. Small hæmorrhages, induration-areas and giant-cells are proof of the death of many. Only a few parasites are able to find a way out of the lungs. The visceral pleura is pierced and the diaphragm entered. This may occur adjacent to the pleural cavity, adjacent to the mediastinum, or adjacent to the pericardial cavity.

Very often an adherent pleuritis diaphragmatica is set up by which means a safe path is established for the cercariæ. In a high percentage of the observed cases this pleuritis was found to be present.

Three days after the infection, cercariæ can be found in the diaphragm. After piercing the diaphragm some parasites reach the body-cavity. Presumably these die. In no section of the free margin of the liver have I seen any penetrating cercariæ. The remainder then reach the liver at the place of adhesive connexion with the diaphragm.

In the stroma of the liver, development proceeds with great speed. After a few days the cercariæ begin to resemble adult parasites. The time of reaching the portal veins appears to vary considerably. The reserve of food (blood) seems to suffice for many days. After eight days parasites can be found in the stroma of the liver, filled with remains of previously sucked blood, but apparently this is the limit. After this the young parasites are found only in the ramifications of the portal veins, where further development takes place.

Only very exceptionally are a few cercariæ found in the arterial circulation. Twice a *Cercaria mansoni* was found in an artery of the hilus of the kidney. Once a number of dead bodies of the unknown species were found in the mesenteric artery. Presumably in these cases a few cercariæ reached branches of the vena pulmonalis, and thence the great circulation.

The observation of Turner, who found in a pulmonary vessel full-grown bilharzia parasites, has to my knowledge never been corroborated by another observer. [Ruffer recorded this.—R. T. L.] My opinion, based upon pathological-anatomical alterations in the lungs of bilharzia patients, is that full-grown parasites in the lungs need not be rare. The important alterations, many times observed by myself in Surinam, cannot easily be explained by emboli alone. Moreover in one case I succeeded in squeezing out of the pulmonary-vessels a few full-grown bilharzia parasites. In cases of severe infections it appears most probable that a few cercariæ, remaining behind in the pulmonary vessels, may completely develop under favourable conditions.

According to the described experiments, the cycle of development of *Schistosoma mansoni* in the final host takes place as follows through :—

(1) *Skin or mucous membrane.*
(2) *Connective tissue* (a few, remaining there, die).
(3) *Veins.*
(4) *Right heart.*
(5) *Pulmonary artery* :—
  (*a*) can seldom completely develop there (?).
  (*b*) can seldom obtain access to the pulmonary veins and so into the general circulation (?).
  (*c*) reach the stroma of the lungs and become aggregated in the base of the lung.
(6) *Base of the lungs* :—
  (*a*) the majority remain there and die.
  (*b*) the remainder pierce the pleura and reach the diaphragm :—
    (i) via pleural cavity.
    (ii) via mediastinum.
    (iii) via pericardial cavity.
    (iv) directly, after an adherent pleuritis diaphragmatica is set up.
(7) *Diaphragm* :—
  (*a*) from there arrive in the abdominal cavity, presumably to die.
  (*b*) reach the stroma of the liver at the point of connexion with the diaphragm.
(8) *Stroma of the liver.*
(9) *Hepatic portal vein.*

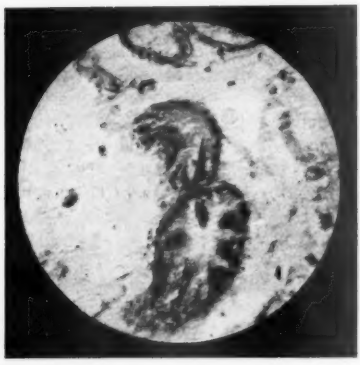

FIG. 1.

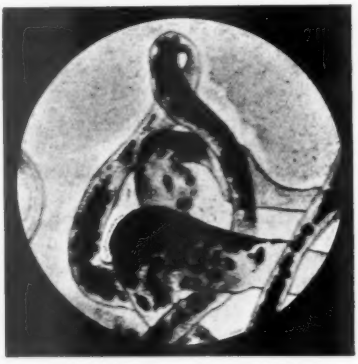

FIG. 2.

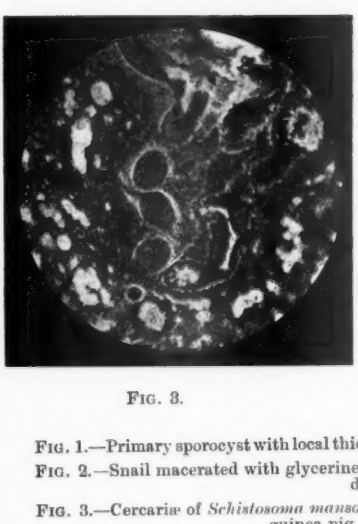

FIG. 3.

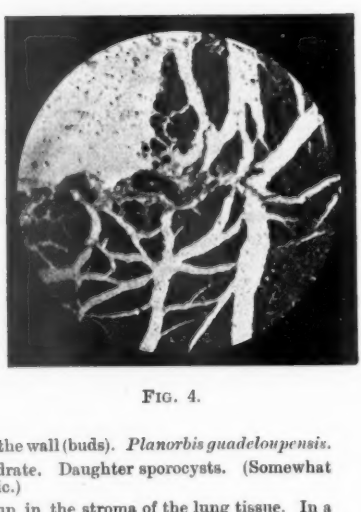

FIG. 4.

FIG. 1.—Primary sporocyst with local thickenings of the wall (buds). *Planorbis guadeloupensis.*

FIG. 2.—Snail macerated with glycerine chloral hydrate. Daughter sporocysts. (Somewhat diagrammatic.)

FIG. 3.—Cercariæ of *Schistosoma mansoni* heaped up in the stroma of the lung tissue. In a guinea-pig two days after infection.

FIG. 4.—Cercaria of *Schistosoma mansoni* penetrating the diaphragm. In a rabbit six days after infection through the skin.

JOINT DISCUSSION No. 9.

Section of Medicine, Section for the Study of Disease in Children, and Section of Dermatology.

Chairman—Dr. HUGH THURSFIELD (President of the Section of Medicine).

DISCUSSION ON THE USES AND LIMITATIONS OF ULTRA-VIOLET LIGHT THERAPY.

Dr. ERNEST DORE.

THE use of ultra-violet light radiation in therapeutics is due to the work of Niels Finsen, of Copenhagen, and his observations on the exclusion of these ultra-violet rays in the treatment of variola and their employment in lupus, in 1893 and 1894, are now classical.

The so-called red-light treatment of small-pox has not, so far as I am aware, been much used in this country. Professor Rasch, of Copenhagen, during a recent visit to London expressed to me his surprise at the neglect of the treatment here.

Phototherapy by means of concentrated actinic rays was introduced into this country in the year 1900, and I will briefly record its progress since that time.

LOCAL APPLICATION.

I shall deal first with the local application of light which particularly concerns the dermatologist, and consider general light treatment in its wider medical aspect later.

Finsen's method of treating lupus was first employed in hospital practice at the London Hospital nearly twenty-seven years ago; Dr. Sequeira's publications of the results obtained in 1900 and subsequent years, and his translation of Finsen's "Phototherapy" in 1901, are well known.

In the same year (1900) the late Sir Malcolm Morris, with whom I had the privilege of being associated, also treated numerous cases of lupus and other diseases of the skin with the original Finsen lamp obtained from Copenhagen, the results, published in 1901 and subsequent years, being similar to if less successful than those obtained at the London Hospital. Finsen's original lamp, with focussed rays, adapted for the treatment of four patients, although still in use at the London and some other hospitals, proved unsuitable for smaller institutions and private work, and was followed by the smaller apparatus constructed by Reyn on the same principle for the treatment of a single patient and called the Finsen-Reyn lamp. This lamp, owing to the small area treated and the long exposures required, was to a certain extent replaced by apparatus giving unfocussed rays, to which shorter exposures were required, as in the lamps of Lortet and Genoud and their modifications, and those devised by Bang and Miller with iron electrodes giving a high proportion of ultra-violet rays. These rays were not concentrated but brought into close proximity to the skin, ice being used to cool the surface and render the tissues anæmic.

At this time light treatment was generally recognized as a valuable therapeutic measure, chiefly in certain local diseases of the skin, but it did not come into use for general purposes until many years later. In the recent popularity to which the treatment has attained, the original work of Finsen, followed by that of Sequeira,

Whitfield, Adamson, MacLeod and others in this country, has, to a certain extent, been over-looked.

In 1916 a considerable impetus was given to actino-therapy owing to the introduction of the Simpson lamp with Wolfram electrodes. Dr. Sequeira demonstrated in regard to these rays what Finsen had shown in the case of the actinic rays of the sun and the arc-light, that they not only caused an acute inflammation of the skin, but that their action was extremely superficial, and that they were intercepted by dental aluminium films, gutta-percha tissue, and even by a thin layer of paper placed on the skin. This fact of the slight penetrative properties of the shorter actinic rays is, I think, sometimes overlooked at the present day. The exaggerated results claimed by the introducer of the Simpson lamp caused considerable discussion at that time. Not only asthma, but a variety of other conditions were stated to be benefited by inhalation of the vapours, and I remember being told by Simpson that a cat frequenting the premises had unexpectedly given birth to a litter of kittens, presumably as the result of constant exposure to the light.

The original Finsen and Finsen-Reyn lamps and the Simpson lamp have now to a large extent been superseded by different varieties of mercury-vapour and tungsten arcs, whilst the larger carbon arc lamps are generally reserved for general light baths. The lamps now employed do not differ greatly from those originally used, and, in my opinion, speaking from the dermatological point of view, although much work has been published since the revival of light-therapy during the past few years (I especially mention that of Dr. Sibley, Dr. O'Donovan, the late Dr. Castle, and Dr. Roxburgh), it is doubtful whether very much of value has been added to the work originally done by those who adopted Finsen's methods twenty-six years ago.

In 1918 and 1919 Axel Reyn, of the Finsen Light Institute, Copenhagen, wrote upon the improved results obtained in the treatment of lupus by general light baths in addition to local treatment, and stated that the percentage of cures was increased from 60 to 80 per cent. when general as well as local treatment was adopted at the outset. Heiberg and Karl With stated the percenage as 90, and claimed that carbon arc-light baths could cure lupus without any other treatment. Dr. Sequeira confirmed the value of the combined method which he introduced at the London Hospital, his results being published in 1923.

General Application.

This brings us to the consideration of light treatment in its more general aspect, which dates back to the years 1902 and 1903, when helio-therapy was employed in surgical tuberculosis by Bernhard. He was followed by Rollier, at Leysin, and since 1908 by Sir Henry Gauvain, at Alton and Hayling Island, who used both natural and artificial sources of light, and by numerous other workers since that time. The treatment of surgical tuberculosis scarcely comes within the scope of this discussion, and I shall pass on to mention the discovery of the value of ultra-violet light in human rickets, by Huldschinsky, in the year 1919. This observation was confirmed in 1920 and 1921 by the English workers in Vienna—Chick, Dalzell, Mackay, Hume, Henderson Smith, and others, by McCollum, in Baltimore, and by Hess and Steenbock, and their co-workers in America.

There is only time to refer to a few of the experiments conducted by these observers. Hume and Henderson Smith showed that air irradiated by the mercury-vapour quartz lamp promoted the growth of rats fed on a vitamin-free diet, and that irradiation of the sawdust used in the cages had a similar effect. Steenbock, and also Hess, found that exposure of various vitamin-free oils and foodstuffs to ultra-violet irradiation invested them with antirachitic properties. Hess also demonstrated that cholesterol became activated by ultra-violet light, and that irradiated calf skin and human skin fed to rats prevented rickets.

I must leave the consideration of the action of ultra-violet light in rickets and tetany, in anæmia and wasting diseases of children, and in glandular and peritoneal tuberculosis, etc., to our colleagues of the Section of Medicine and the Section for the Study of Disease in Children; but there is a point I should like to mention here relating to the general effect of exposure to light. It is a common experience among operators that a feeling of stimulation results from treating patients with ultra-violet rays. Nurses and attendants often notice the beneficial effect on their health, in the way of the prevention of catarrh, etc. It seems possible, therefore, that ionization of the air and the liberation of ozone or of some other substance may produce such effects independently of the actual light radiations. The improvement said to follow in asthma and catarrh, the fact often observed that treatment of one part of the body will also benefit the whole organism (e.g., Hess noted that treatment of one epiphysis in a rickety child led to improvement in the opposite limb), favour this supposition, although, of course, the effect may be explained in other ways. The experiments of Haxthausen are interesting in this connexion. He found that anti-rachitic substances, such as cod-liver oil and inert vegetable oils which had been irradiated, would affect a sensitive plate after some hours' exposure. This effect could not be due to ultra-violet irradiation because it was stopped by the intervention of a quartz plate, nor to radio-activity, since cod-liver oil could not discharge an electroscope. Haxthausen also found that the skin fat of persons washed with ether and evaporated in a dish produced the effect on the plate only in previously irradiated skin, thus indicating a chemical effect on the surface skin fat. Sonne, who quotes these experiments, says that if the effect is not due to a genuine radiation, it is better to try to describe it by the diffusion of some substances in a vaporous form.

The Injurious Effects of Light.

No discussion on light treatment would be complete without consideration of the harmful effects that may be produced by it. It is unnecessary to do more than mention the pathological conditions associated with constant or excessive exposure to the rays of the sun, such as solar dermatitis, hydroa æstivale, xeroderma pigmentosum, senile keratosis, etc., while erythema ab igne is due to the heat and infra-red rays at the opposite end of the spectrum.

Certain mechanical dangers may be mentioned, such as breakage of quartz burners, which happened in my practice on one occasion, the danger of burns from spluttering electrodes, the risk of electric shock, especially when lamps are installed in bathrooms, the deleterious effect on the eyes, and also the risk of a burn from excessive exposure, as in a case reported by Dr. MacCormac and Dr. McCrae. Added to these are the debility and depression produced by too frequent or too lengthy applications, the possibility of lighting up a general tuberculosis or aggravating a febrile disease, and so on. With regard to other contra-indications, such as glycosuria, I cannot speak from personal experience, and such points as the effect on metabolism and on the blood-serum and blood-cells, also the vexed question of the necessity or otherwise of producing erythema and pigmentation, I must leave for discussion.

Lastly, I should like to suggest some points for consideration:—

(1) *From the Dermatological Point of View.*—Is the inflammatory reaction we aim at producing in lupus, alopecia, etc., a specific reaction? and if not, can equally satisfactory results be obtained by other and simpler measures? Is the benefit claimed in certain cutaneous affections, e.g., acne and psoriasis, due to the indirect rather than to the direct action of light, such as exfoliation of the epidermis, increased sweating, etc.?

178 Oddy: *The Uses and Limitations of Ultra-violet Light Therapy*

(2) *From a General Standpoint.*—Can the results obtained by ultra-violet light in malnutrition, rickets, tetany, tuberculosis, etc., be equally well produced by the administration of cod-liver oil, by appropriate diet, fresh air and improved hygienic measures? In other words, are the claims now made for ultra-violet light treatment in the lay and medical press, and its increasing advocacy as a public health measure, justified by the results obtained?

Dr. H. M. Oddy.

I propose to deal with the matter under three headings: (1) The differences in reaction as between adults and children, and differences in technique and dosage; (2) those biological effects of light which are of special interest in children's diseases; (3) the uses and limitations of light-therapy in children's diseases.

As a rule, children react much more easily and quickly to light than adults do, hence the dose must be carefully adjusted. Dr. Humphris has suggested that for a child the dose should be half that for an adult, for an infant half that for a child; a very good rough rule. As the skin of the infant is thinner than that of the older child and the adult, it might be supposed that erythema would ensue more easily in children on the application of light, but that is not the case. I believe that Dr. Eidinow was the first to point out that the production of erythema in infants does not follow the usual routine; for some unknown reason erythema is more difficult to produce in the infant. This is important, because it means that the minimum erythema dose cannot be used as a guide in treatment.

The question of overdosage is especially important in the case of children, because the adult can usually clearly describe his feelings and experiences, whereas the child and infant cannot, therefore one has to rely on signs and symptoms observed by oneself. The following I regard as important signs of overdosage: (1) Increased irritability and disturbed sleep; (2) persistent loss of weight or failure to gain weight while under treatment, with, possibly, digestive disturbance and loss of appetite.

In children the best results seem to be obtained with the mercury-vapour lamp except in the case of surgical tuberculosis. I have no experience of the effects of general baths with the tungsten lamp, and I shall be glad to hear views on this from other speakers. I have used the tungsten lamp for local applications.

The course of treatment varies with the disease being dealt with. One should beware of continuing the treatment too long; twelve, fifteen or twenty treatments at the rate of two or three a week should be followed by rest for a month or two, after which, if considered necessary, a further course could be given.

With regard to biological factors, time does not permit of my entering into these in detail, but there are one or two points I should like to mention.

The question of pigmentation is still debated, and it has been pointed out, by Dr. Gamgee and others, that in small infants pigmentation does not, as a rule, occur. Still, in spite of that, the clinical results from the treatment are excellent, an experience with which most observers agree. Among the children of all ages whom I have treated, both in hospital and privately, very few have shown marked pigmentation, yet the clinical results have been decidedly good. I doubt whether the statements in the books as to the value of pigmentation are correct.

The question of the effect of the rays on cholesterol is very interesting. If cholesterol is exposed to ultra-violet light, it becomes in some way chemically altered, so as to be antirachitic. Skin, removed from an animal and similarly rayed, shows a like property. When the body is rayed the cholesterol in the skin is altered and becomes antirachitic. Experiments on the blood have shown that when the calcium and the phosphorus content of the serum is low, it can be raised to the

normal by means of light baths; but if it is normal to begin with, it cannot be raised above normal by radiation. In most cases of rickets the serum phosphorus is low, and in tetany the serum calcium is low, and these can be raised to the normal by means of light. The experiments on cholesterol are important in another way, in that they open up a very interesting field for speculation and research. Probably cholesterol is not the only substance in the skin which may be acted upon by light; probably other substances are so activated and enter the blood, thus exerting their effects on different organs. It is known, for example, that the acidity of the gastric juice can be altered during treatment by light baths.

Metabolism is much improved by light baths, but Professor Leonard Hill has shown that this is mainly due to the effect of cold air on the patient. Still, it must be remembered that when the patient, stripped of his clothing, is receiving the rays, both light and air impinge on the skin together.

I hope some one will speak about basal metabolism. I do not think much has been recorded as to changes in the basal metabolism resulting from light baths.

Concerning the clinical uses of light, so much has been written and spoken about rickets that I do not propose to enlarge upon that point now. There can be no doubt that light is one of the best methods of curing rickets, though that disease is cured equally well by means of cod-liver oil, and probably as well by giving irradiated cholesterol. I would therefore say that light is not to be regarded as taking the place of these other methods; it must be used in conjunction with them.

In surgical tuberculosis—especially in such diseases as tuberculous peritonitis and glandular tuberculosis—light is an indicated treatment and does good, but an important exception must be made where active disease of the lung is present. When the lungs are only slightly affected and there is no pyrexia, it is safe to employ light treatment, but the dose must be carefully graded. On the whole, bone and joint cases do better under light than do peritoneal and glandular cases which show only a slow response to the treatment, though they do respond in time. Usually the type of peritonitis which is accompanied by effusion does less well under light than does the dry form. The treatment must extend over a long time, and that is a difficulty, especially when patients are living at home and come to hospital at intervals for the applications. It is important to realize that light ought not to take the place of other treatments; it must be used in conjunction with them, and surgery, when necessary, must be included. One of the features which strike one in connexion with Rollier's clinic is the length of time the patients are under treatment; no surgery is employed to separate the sequestra; they are left to separate naturally under the light. I hold that surgery may sometimes shorten the treatment.

Light applications are also useful in pregnancy and especially in lactation, and very good results have been obtained in the treatment of nursing mothers by this means. If a mother's milk shows signs of failing, treatment by light applications should be given to her. Good results likewise follow its use in malnutrition of infants, also in some cases of marasmus; but very severe cases of marasmus which do not respond may be harmed by the light; in addition, I do not like to strip the marasmic child to expose it to the radiation. Light is especially good for restless, screaming children who sleep badly, and for those who do not react to dieting. Such children become quieter, digest their food better, and begin to gain weight.

There is also the type of child which Dr. Donald Paterson has described under the name "the hypotonic child." This type does not show any evidence of organic disease, but is thin, pale, and tired, restless and listless, with flabby, hypotonic muscles. In such cases light is essential, and as a rule the response to it is excellent. Still, it fails in them more often than one would expect. In some cases in which there is failure, I think it is either because too large a dose has been given at first, or

180 Eidinow: *Uses and Limitations of Ultra-violet Light Therapy*

because the doses have been increased too quickly. Moreover, in out-patient practice one cannot regulate the home conditions, and the child may be living in a bad environment and may be receiving the wrong food.

Sir Henry Gauvain's and Dr. McCrae's observations as to the benefit of sunlight to children who, without being actually mental deficients are mentally backward, are very interesting, particularly, perhaps, from the point of view of education.

In the debility which follows operations there is no tonic which has such a good effect as light baths, both for children and adults. Light is excellent for treating paralysed muscles, especially in infantile paralysis; it tones up the muscles and improves the nutrition and circulation. Dr. Murray Levick uses screens which cut out much of the heat and allow passage to the red-light rays, and he claims that this enhances the good result.

Some cases of asthma, particularly if associated with bronchitis, show an excellent response to light baths carefully graduated.

Blood diseases, especially forms of anæmia in which the hæmoglobin is low, could be treated by light. I have not had experience of treating leukæmias in this way, but, theoretically, light seems to be contra-indicated in these cases. Dr. Punch and Dr. Wilkinson recently published a case of Hodgkin's disease, in which light yielded temporary benefit, but not much could be expected from it in such a disease as that.

I have purposely refrained from mentioning too many diseases in this connexion, because anyone who has read the English and American literature of the last five years may be pardoned for thinking the age of miracles has returned; other physicians seem to believe that light treatment is only a fad. Both are wrong, for in conjunction with other methods it is certainly a valuable addition to our methods.

Dr. Albert Eidinow.

A correct summary of this question is too lengthy a problem, therefore I shall confine my remarks to a consideration of the scientific and biological sides of the light question, leaving to others the discussion of the clinical problems. Ultra-violet rays, or that portion used in the treatment of disease, consist of rays extending from 3,500 to 2,500 Angström units in length. It is this group which has been shown to have biological and therapeutical effects. When searching for a medium—drug or vaccine—one looks for a substance which may, in itself, have a direct bactericidal effect, or a substance which will produce such changes in tissues or blood as will directly, or indirectly, activate bactericidal action without causing excessive damage to normal tissue. Can light be said to possess any of those properties? From research which has been carried out at our institute, we were able to prove definitely that light could increase the bactericidal properties of the whole blood, tested *in vitro*.

What factors in the tissues are responsible for this change? Is it the direct action of light on the skin? Experiments with living skin, dead skin, and irradiated skin in comparison with normal skin, show that no direct bactericidal substance is produced in the skin as a result of irradiation.

Is it a direct action of light on the blood? Here one is faced by a problem. The penetrative power of ultra-violet rays is small, hence one cannot well conceive of these rays having any direct effect on blood-corpuscles. I have been able to show that when the blood is exposed to light the bactericidal property of that blood is destroyed, but when that blood goes back into the circulation the bactericidal property of the whole blood is increased, when tested *in vitro*.

Can it be conceived that, when light is applied locally to a diseased lesion, such as a patch of lupus, the tubercle bacilli at it are directly destroyed by the light? Workers within the Finsen Clinic say that the rays penetrate to the lupus nodules and produce a direct bactericidal action.

Ultra-violet rays are bactericidal, when one tests bacteria—staphylococcus, streptococcus and tubercle bacillus—in a simple fluid such as saline. The same organisms suspended in blood-serum or in defibrinated blood are much more difficult to kill on exposure to light. An exposure of tubercle bacillus in blood-serum or defibrinated blood to two mercury vapour lamps at 8 in., and kept cool by water, does not destroy the bacilli, even after one hour's exposure. This has been determined by inoculation of this irradiated mixture into guinea-pigs.

Next, one comes to the problem of the action of light on the skin, or on tissues beneath the skin. Can the light produce some substance which can be absorbed and carried through the circulation, setting up a bactericidal effect? The results of these experiments are not yet complete. When sections of irradiated skin are examined, microscopically, at the stage at which erythema has been produced and œdema is present, it has been found that, surrounding the smallest capillaries and lining the swollen endothelium, there are leucocytes clinging to the sides of the capillaries. One can see diapedesis of the leucocyte wandering through the capillary wall in the subcutaneous tissue. An erythema dose of light produces a "fixed" leucocytosis beneath the skin.

This gives a possible clue as to the biological action of light. Light, acting either on the skin or on the capillary blood-vessel, i.e., superficial blood-vessels, attracts leucocytes and produces changes in the wall of such vessels, so that permeation of plasma through the capillary wall occurs, also diapedesis of leucocytes in the œdematous tissue.

The foregoing summarizes the research as far as my colleagues and I have carried it on the biological side.

In dealing with the question of rickets, credit is due to Rosenheim and Webster, who have made great strides in the solution of this problem. They have been able to show that the irradiation of ergo-sterol is probably the factor in the cure of rickets. At first this substance was thought to be cholesterol, but these workers found that ergo-sterol was combined with cholesterol. The irradiation of this substance was found to be the most potent factor in preventing rats fed on rickets-producing food from developing the disease. It is thus easy to see that a small irradiation of the skin will activate these sterol substances, and that the absorption following will prevent the occurrence of rickets. It does not seem necessary to be told much more in order to see how light acts in this disease.

With regard to the treatment of the skin with light, and the technique of exposure, following on the local treatment of lupus by Finsen and his colleagues, one arrives at the stage when general irradiation of the body is employed. Careful investigation has shown that the skin rapidly becomes immune to the action of ultra-violet rays. Directly after the first exposure of a patch of skin to a minimal or subminimal dose of light, that patch becomes immune to radiation, and so remains for fourteen days, i.e., until the stage of desquamation has passed and young skin has developed.

When we were investigating the bactericidal effects of light we were able to show that the surface area of skin exposed to the action of the rays was an important fact in determining whether the state of that blood would improve, or remain stationary, or get worse after exposure; and we were able to demonstrate that there was a definite optimum area of skin to expose in order to secure the best bactericidal effect on the blood. That area is about one-sixth of the body surface, such as the chest, or the back,

182 Gauvain: *Uses and Limitations of Ultra-violet Light Therapy*

or the front of both legs. The patient was made to rest fourteen days before the exposure was repeated. We worked on the criterion of trying to obtain a minimal erythema with each exposure to the rays. When this technique is being carried out it has been found unnecessary to employ large massive exposures; the patient can be kept sensitive to the rays for a longer time on short exposures. This matter requires much more clinical investigation than my co-workers and I have been able to give it. It may alter the present technique of exposure to the sun's rays, and to arcs and mercury vapour lamps. If what I have said is true, it is possible that many of the bad and indifferent results obtained with the rays have been due to the fact that the patient was not getting the effect of them at all, as the skin was immune.

I will conclude with some remarks on dosage, overdosage and dangers from light. The question of dosage depends much more on the area of surface of skin exposed than on the actual quantity of light employed. A minimal erythema dose, a full erythema dose, and a blistering dose, will all have the same effect, if one does not expose too large an area. The chief point, in the matter of contra-indication for such applications, is the degree to which the disease has progressed. In conditions of acute septicæmia and acute phthisis, with the blood in a very impoverished state, an excessive exposure to the rays, without a careful check on the bactericidal powers of the blood, may be very dangerous.

Sir Henry Gauvain, M.D.

I prefer, so as not to occupy too much time, to limit my remarks to effects of light therapy in surgical tuberculosis. I would point out that though this discussion was on the effects of ultra-violet light, yet pure ultra-violet light, unmixed with other forms of radiation, has not been so far mentioned. Indeed very little work has been done on the use of pure ultra-violet light alone. Pure ultra-violet rays can be used by placing before a suitable lamp a piece of Wood's glass, but this has attracted little attention. The method I have mentioned has been used in certain cases, such as for diagnosing ringworm of the scalp, in which condition pure ultra-violet light gives a beautiful fluorescence.

What is the effect of the other rays which are employed? In actual sun treatment, which I still regard as the best form of radiation treatment of surgical tuberculosis, the proportion of rays is: infra-red 80 per cent., visible rays about 19 per cent. ultra-violet light only about 1 per cent. In the carbon arc there is more ultra-violet light, but these lamps are not so good as pure sun baths. With mercury vapour there is more ultra-violet light still, and a considerable number of rays which do not occur in the solar spectrum. Yet, looking at the practical effects of general light treatment, the value of these different lights diminishes in surgical tuberculosis in proportion as the ultra-violet light increases.

I am aware that biological investigation has shown certain effects which have been largely attributed to ultra-violet light, mixed rays having been used, but I submit that there is clinical evidence opposed to the view that the benefits of light treatment are entirely due to ultra-violet light. And if the value of treatment be estimated by certain specific effects of ultra-violet light, for example the work done by Eidinow, who has demonstrated the hæmobactericidal properties of light, then we are in the false position of basing our treatment on one biological response, whereas actually many other reactions may be simultaneously taking place. Is it wise that only one reaction shall be used as a measure? It is a point of great practical importance.

If what I have said is correct, namely, that pure sunlight is the best form of treatment, surely there must be a great deal in those other radiations which are not ultra-violet rays. It is known that the visible rays have a much greater penetrating

property, that they get through the skin and are absorbed into the blood-stream and have thermal effects. The infra-reds are absorbed on the surface because they are absorbed by water, the unpleasant burning resulting probably limiting their utility.

In the treatment of surgical tuberculosis I do not regard light as in any way a specific treatment; I agree with what previous speakers have said, that light treatment should be regarded merely as an aid—though a very valuable aid—to other treatments; it should supplement, not supplant them.

Does light treatment help? On clinical grounds, yes. It is only necessary to see, in a country hospital, how sunlight accelerates recovery; there is indisputable evidence that the results are best when there is the best light value. In winter months the speed of progress of patients is not so good, but can be accelerated by employment of artificial light. Dr. Oddy has mentioned the important effect of the impingement of cold air on the bodies of these patients, and Professor Leonard Hill and Dr. Campbell, working at Alton and Hayling Island on this interesting subject, have shown conclusively that the basal metabolic change in those undergoing sunlight treatment is due not to exposure to the sun, but to exposure to cold air.

Another important practical point is, that though there may be certain biological effects following exposure to light—for instance, cholesterol effects, skin effects, and increase of hæmobactericidal power—yet by giving a similar dose of light to twelve different patients undergoing treatment for surgical tuberculosis, different effects are observed in each. The effects I have mentioned do not, in themselves, have a very direct bearing on the improvement which may be expected in the patient, and it has been forced on me, in a way which cannot escape attention, that the value of light depends largely on the result of exposure to light and the general reaction which follows rather than to specific responses. One type of reaction which I regard as of great importance is pigmentation of the skin. This is a response to a light stimulus, and without being able to give very definite scientific reasons, many clinicians strongly consider that the majority of pigmenters do better during treatment than most non-pigmenters. It illustrates power of response in the individual. All have their basal metabolism increased by exposure to cold air, and improve by dietetic and hygienic measures, whether pigmenters or non-pigmenters, but they do not respond to light in the same way. If a number of patients are taken it is found that the length of treatment required is greater in those who do not show a response to light than in the remainder. The effect of light is to produce a very complex reaction, one made up of many constituents, and it is the sum total of these which produces the helpful or harmful effects in light treatment of cases of surgical tuberculosis. And these remarks hold good for all ordinary conditions being treated, except conditions such as rickets, where light has a specific action.

In what way does light help in general treatment in cases of surgical tuberculosis? I think there is no doubt that the healing of lesions is accelerated. In cases in which the disease is still advancing and decalcification proceeding, it is necessary to wait until active advance ceases before restoration occurs. The progress of the disease may be lessened by light, but it occurs only when healing commences.

A little boy, who has now been with me eighteen months, arrived with acute disease of the hip-joint. The X-rays showed perfect formation of head and neck of the femur, and a small abscess appearing. He was cachectic, a freckler, and a non-pigmenter. He has been given careful exposure to light and associated general and orthopædic treatment, but the disease is still progressing. The head of the femur has almost disappeared and the neck has been largely absorbed, in spite of his having had the appropriate treatment which such patients are given at the institution.

Therefore, light treatment is not specific; that boy has not the right power to react to light. In cases which respond well to treatment by light, which pigment and feel all the better for exposure, cure is undoubtedly hastened.

184 Sequeira: *Uses and Limitations of Ultra-violet Light Therapy*

As an example of the beneficial effect of light therapy, Dr. Oddy has mentioned the effects which Dr. McCrae and he investigated as to the mental improvement which manifested itself in patients. Abscesses frequently, but not invariably, tend to form pus less quickly, and heal more rapidly under the influence of light.

A non-pigmenting spinal patient whom I could not expose to general sun treatment had a double psoas and lumbar abscess, which required many aspirations, and as soon as she was put into ordinary sunlight she felt ill, and had to be removed. Over eighty aspirations were needed. Under very careful carbon-arc treatment there has been a remarkable tendency for the abscesses to heal, and only two aspirations have since had to be performed.

The healing of sinuses is often hastened by light, and generally healing occurs with very little scar formation.

Sequestra are sometimes painlessly extruded, and many other effects may be instanced which considerations of time prevent me from detailing.

With regard to the direct action of light on lupus, Dr. Eidinow opposed the original statement that light had a direct lethal action on the tubercle bacilli in lupus lesions; but I do not know that he is altogether right. Lupus nodules themselves are more easily permeable by light than the normal surrounding skin. A simple experiment can be performed on a large area of lupus vulgaris which is not ulcerating. If that area is exposed to a brisk local dose of mercury vapour light, it can be seen that wherever there are nodules there are little blebs forming, while the rest of the skin has not been attacked sufficiently to blister. There has been greater penetration and greater response in the nodular areas than in the surrounding skin.

Dr. J. H. Sequeira.

One or two questions have been raised in this discussion which I think can be answered.

In the first place, I fully agree with Sir Henry Gauvain as to the utility of mixed rays. In all the clinical work which has been done in this connexion up to the present, mixed rays have been used. I also agree that among sources of the light used the sun comes first, then the carbon arc, perhaps reinforced by tungsten, and lastly the more limited mercury vapour radiations.

The first point I wish to make is, that in my own clinic general treatment of the human body by light has, without any local measures, healed lupus vulgaris of the skin and mucous membranes. This was first learned from the work of Strandberg, Heiberg, and within the Finsen Clinic [1, 2]. But I agree that in no case is it wise, even in the treatment of a disease like lupus, to rely entirely upon general irradiation of the body.

A question has been asked as to whether any work has been done on basal metabolism. Professor Leonard Hill was good enough to send Dr. Argyll Campbell to work in my clinic to study this point, and the basal metabolism of patients was estimated before and after irradiation. Cases were taken which had not had any light treatment, and the result of the observations was that no change in the metabolism was produced solely by the general light bath. In the London Hospital clinic it has been found, as a result of four years' work, that the giving of light baths does not prevent the onset of acute specific fevers; our patients have suffered from varicella, measles, whooping-cough, and even cerebro-spinal meningitis. But it must be admitted that in post-febrile debility great benefit is derived from general light treatment. An experimental investigation is now being conducted in a Metropolitan Asylums Board institution with regard to whooping-cough, on the suggestion of the Committee of the Medical Research Council, which is dealing with the biological action of light. It is proposed to irradiate every alternate patient in that hospital, and it is hoped that helpful data will be obtained.

In the discussion the question of pigmentation has been raised, and considerable divergence of opinion as to this matter exists. I have seen extraordinarily good results in the treatment of extensive lupus in patients who have not pigmented at all.

With regard to the hæmobactericidal action: I have no practical experience of this, but I understand that extraordinary care is necessary. Were the observations made on staphylococci?—[Dr. EIDINOW: On staphylococci, streptococci and pneumococci; mostly on staphylococci.]—The variation between normal reaction and abnormal reaction is so small that large experience and careful observation are needed to ensure accurate results and records.

There is also the question of the area exposed to the light. Positive statements have been made as to this, but here again my view is that more and very careful observations are needed. Some of my cases have been treated in this piecemeal way, and I have been so disappointed with the clinical results that I have been forced, by the opinion of the workers in my department, to put the patients on to more general and larger exposures. The results in limited exposures are not, in my opinion, worth the trouble taken, and not as good as those from general bath exposures.

My last point relates to a matter which has not yet been touched upon in the discussion: the extraordinary value of general light baths in the treatment of some of the more chronic affections of the eyes, especially in children. Mr. Goulden, Ophthalmic Surgeon at the London Hospital, some years ago asked me to treat a number of his patients from that hospital and from the Royal London Ophthalmic Hospital, Moorfields, by light baths, especially cases of phlyctenular conjunctivitis, corneal ulcers, and some cases of tuberculous iridocyclitis. The success obtained was so great that a lamp for the purpose has been introduced into the Royal London Ophthalmic Hospital, and Mr. Duke-Elder, who is in charge of it, has written a paper stating some remarkably good results [3].

The great benefits of the treatment in certain cases of debility in children are probably due to the removal of some chronic infection.

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Dr. KATHERINE GAMGEE.

I agree with practically everything Dr. Oddy has said as to the effect of this treatment in children. In connexion with children it is necessary to remember that the child below 4 or 5 years of age reacts differently from the older child whose reactions resemble those of the adult.

In regard to rickets, which has been mentioned during the discussion as responding quite as satisfactorily to cod-liver oil and good hygiene, I think that the time taken by the treatment is an important factor. I have seen numerous cases of severe rickets making, apparently, no headway under cod-liver oil, go rapidly forward when put under light treatment. It must be remembered, too, that many children cannot take cod-liver oil.

Among the cases which I consider especially suitable for ultra-violet light therapy (such as the case of the adult or child convalescent from acute disease or after an operation), the nervous child reacts to suitably applied light therapy in an extraordinary way. I have treated a very large number of cases of nocturnal enuresis, and though I have only analysed a small series of about thirty consecutive cases, I believe that this form of therapy shows a slightly higher percentage of cures

186 Gamgee: *Uses and Limitations of Ultra-violet Light Therapy*

than any other in similar cases. I recognize, however, that it is difficult to draw accurate conclusions from so small a series of analysed cases.

The convalescent child or adult, especially the case about to be submitted for operation for tonsils or adenoids, is particularly suitable for this form of treatment, whilst those suffering from rickets in poor districts and from anæmia, and from malnutrition, and the subjects of "pre-tuberculous" states among all classes, will always form a large proportion of the suitable cases in children. I think the greatest possible care is needed in treating cases of possibly latent tuberculosis, and the treatment is usually contra-indicated, except under expert sanatorium supervision, in cases of active tuberculosis.

I have found the results obtained in anterior poliomyelitis to be disappointing unless the treatment is begun early.

With regard to the area of skin irradiated, it is better in my experience to irradiate fairly large areas of skin each time, but I agree with Dr. Eidinow that it is most important, if the best results are to be attained, to keep the skin "light-sensitive" and that it is better not to irradiate the same area of skin at consecutive treatments.

Early in 1924, I observed that the patient soon become "stale" and ceased to give the maximum response to light unless the course of light treatment were intermitted. This seems one of the most remarkable points in artificial light therapy and one about which nothing appears to be mentioned in books, though I have no doubt that other observers must have noticed it. The effect seems to be independent of the phenomena of pigmentation. It is possible to note an optimum improvement in many cases, with little resulting benefit on prolongation of the course of treatment.

The best results are usually attained by short courses, of a month or six weeks (twice—or thrice—weekly doses), followed by rest and a subsequent course if necessary. I have observed what appears to be a definitely cumulative action, resulting in marked after-improvement in many cases, and I regard the interruption at the right time of a course of treatment as of great importance in obtaining good results. So much so, that I believe that one day we shall discuss "shade treatment" as well as "light treatment." Artificial light can be regarded as a stimulating drug of which the body cells tire rather easily. It is also easy to overdo the actual light dose. It must be remembered that Dorno has asserted that the average high-efficiency mercury vapour lamp is six times as strong in ultra-violet radiation as the noonday high mountain sun. I think it is a mistake to give daily doses over any length of time in the case of the more powerful lamps.

I have had brought to me a small child who had been attending a hospital where she had been receiving daily doses over a period as long as twenty weeks consecutively. The parents were astonished that the child had failed to improve. As soon as the light treatment was stopped the child made headway, and there is little doubt that she had been suffering from over-dosage.

Cardinal signs that benefit is being obtained in any given case are the following:—

(1) Increase in body weight (but this only if it has been below normal).

(2) Increase in hæmoglobin (again only if it has been below normal).

(3) Increased capacity for sleep and more peacefulness with diminished irritability.

(4) The fact that general bodily metabolism is raised *indirectly* as the result of the increased appetite and bodily activity brought about by the tonic effect of the light.

The marked effect obtained in certain nerve cases is especially interesting. The apparently selective action of ultra-violet rays on cholesterol seems to open out large

fields of research. The treatment of a certain number of mentally deficient children exhibiting great restlessness has shown that the restlessness has appeared to be definitely influenced by means of ultra-violet rays and the nutrition improved. A large amount of clinical research will be needed before any definite conclusions can be reached as to countless problems connected with the premature infant, the expectant mother, the mentally dull and backward child and the adult and insane patient, in connexion with this very interesting form of therapy.

Dr. A. C. Roxburgh.

It is almost impossible to make a fair comparison of the values of real sunlight and the artificial forms of light used for treatment, as sunlight must be given in the open air, whereas the carbon and other forms of arc lamp must be used in a more or less stuffy room, as any draught upsets the arc.

It has been said this evening that treatment by light is non-specific, and that is curiously borne out by an analysis of statistics of cases treated at St. Bartholomew's Hospital during twenty-one months, numbering 272.

The curious thing is that whether one takes tuberculous infections, non-tuberculous infections, or non-infective conditions, the proportion of cases in which there was cure or much improvement is round about 21 per cent. in each group, being 20 per cent. for the tuberculous, 21 per cent for other infections, and 22 per cent. for non-infective conditions. In the cases in which there was only some improvement the proportions are somewhat different, being 36 per cent. for the tuberculous, 57 per cent. for other infections, and 48 per cent. for non-infective conditions.

Of all the 272 cases treated, representing fifty-eight different diseases, in 21 per cent. there has been cure or much improvement, in 46 per cent. some improvement, and in 33 per cent. no improvement, or deterioration. Most of the cases were treated with the carbon arc, some with the mercury vapour lamp and some with both.

It is necessary to emphasize the danger of this treatment lighting up an unsuspected phthisis.

The condition of one of our patients at St. Bartholomew's Hospital with lupus vulgaris of the upper lip cleared up dramatically under carbon-arc treatment but a month or two later this patient returned to a medical ward with pleurisy and not long afterwards was sent to a sanatorium with phthisis.

Another case was that of a woman with multiple sarcoids which cleared up remarkably under carbon-arc light treatment. She began to lose weight and was found on examination to have signs of phthisis, though these had not been present when she was admitted to the ward before the light treatment was begun.

Ultra-violet light from the carbon arc or mercury vapour lamp is said to be good for seborrhœic dermatitis. At St. John's Hospital, where I have charge of the carbon arc for in-patients, I have had to discontinue the use of the lamp for seborrhœic dermatitis, because the state of all these patients was rendered worse by it.

Psoriasis is said to be successfully treated by the carbon or tungsten lamp. I have treated a number of cases by this method, but though the condition eventually cleared up under the treatment it took much longer than under treatment with chrysarobin ointment.

Mr. L. V. Cargill.

I am able to confirm Dr. Sequeira's remarks as to the good effects of the carbon arc lamp in cases of phlyctenular disease, strumous keratitis and other tuberculous eye diseases. I deplore the campaign of publicity in the lay press which has aroused futile hopes in the minds of patients suffering from such conditions as detached

retina, choroido-retinal atrophy and glaucoma absolutum. The ocular benefit is derived from a general bath of irradiation, the eyes being protected from any risk of cataract by suitable goggles.

Dr. EIDINOW (in reply).

I would like to add a word as to the differences between sunlight, the carbon arc and the mercury vapour lamp. Though it may be true, as Sir Henry Gauvain has said, that in sunlight there is only 1 per cent. ultra-violet rays, the intensity of that 1 per cent. must be borne in mind; it is greater than can be introduced into any form of artificial light. It is the small region of sunlight represented by the ultra-violet rays between 3,200 and 2,970 Angström units which is answerable for the production of erythema, pigmentation and the therapeutic effects observed. In the experiments in which I have been concerned it has not been possible to produce ultra-violet rays *per se*; they cannot be obtained apart from the visible rays and heat rays, and in our work we are only able to proceed by a process of elimination. We can cut out groups of ultra-violet rays by using filters. When ultra-violet rays are cut out we are unable to produce the biological effects which have been already described.

Sir HENRY GAUVAIN

(in answer to Dr. Eidinow's further remarks) said, I do not think it is so much a question of intensity of the ultra-violet light emanating from the sun, as of the power of response of the individual treated. The very intense sunshine obtained in this country at midsummer is not necessarily the best for treatment. The value of the light used depends to some extent on the admixture of the different rays, but particularly on the patient's power of response.

Dr. ODDY (in reply).

I agree with Sir Henry Gauvain that people talk loosely about admixture of rays. An interesting experiment has been carried out in America by Hess and Unger, which proves that there is a seasonal variation in the amount of serum phosphorus, the quantity rising in the summer, when the ultra-violet part of the solar spectrum is coming through more intensely. This seems to show that it is the actual ultra-violet rays which are concerned in the cure of rickets.

JOINT DISCUSSION No. 10.

Section of Odontology and Section for the Study of Disease in Children.

Chairman—Mr. J. B. PARFITT, L.R.C.P.Lond., M.R.C.S., L.D.S.Eng.

DISCUSSION ON ORAL MANIFESTATIONS OF SYSTEMIC DISEASE IN CHILDREN.

Mr. A. T. PITTS, D.S.O.

THE subject of this discussion is one that covers a wide field and there is a risk that it may cover so large an area as to become unmanageable. On the other hand, the subject is in the nature of an experiment and this discussion may be regarded as being in the nature of a test to ascertain how far it may prove profitable. On this occasion, therefore, it may be wise to allow the course of the discussion to ramble widely, so as to find out its possibilities, and then on some other occasion it may be possible to initiate a discussion on narrower lines. We, as dentists, may properly adapt the saying of Terence that he considered nothing human alien to his interest and say that nothing concerning the mouth is foreign to our interest, whether it be connected with the teeth or jaws, or apart from them. The dentist with his narrow specialized training tends to regard the mouth from the standpoint of the teeth only, and in so doing sometimes overlooks the fact that conditions affecting his special province may be due to non-dental causes. On the other hand the medical practitioner confronted with the more unusual sequelæ of dental disease, even sometimes with its common sequelæ, may be handicapped by a lack of knowledge of dental pathology. He may overlook a dental cause for some oral condition because it is not obvious to him, though here the dentist would be on familiar ground. Each can learn from the other, though perhaps we as dentists have more to learn from the doctor because the range of non-dental conditions which may lead to oral manifestations is so great as compared with the various sequelæ, common and rare, which may result from dental disease. If it were possible to pool our knowledge and experiences I am certain that pathology, dental and general, would be the richer.

I have no intention in the time at my disposal to attempt a catalogue of the various oral manifestations due to systemic disease, but rather to pick out some of them which either have come under my notice or else have excited my interest. I have no confidence in my ability to make any definite contribution to your knowledge but I feel quite certain that I can propound some queries which, if they can be answered, will fill up gaps in our knowledge. I regard myself therefore as being chiefly one who asks questions, and in doing so I may perhaps help to map out the lines of the discussion for those who may feel a little doubtful what we of the Section of Odontology had in mind in suggesting this subject for joint discussion.

Osteomyelitis of the Maxilla in Infants.

The first subject to which I want to refer is the condition described as osteomyelitis of the maxilla. I have seen several examples of this interesting condition at Great Ormond Street Hospital for Children, nearly all in infants ranging from a few months up to two or three years. There would appear to be some doubt as to its ætiology, as to whether it commences as an infection of the antrum or is a true osteomyelitis; and there has been a good deal of literature written upon the subject, especially in the

French journals. Some of these cases come under the notice of the surgeon or laryngologist but others may drift into the dental department on account of sinuses appearing in the mouth. Whatever its origin the condition may assume an aspect which brings it within the scope of dental surgery. The maxilla in infants is little more than a thin shell containing the developing permanent teeth and a small antrum. It is obvious, therefore, that very soon such a condition must involve the crypts of the developing teeth. One might expect that with the thin shell of bone and the numerous bony crypts containing the permanent tooth germs the necrosis would be extensive and dangerous. Yet one finds that necrosis, though often prolonged, is not usually extensive and tends to become limited. Whatever be the explanation of the condition it must be evident that it cannot be primarily dental, for it may commence in a baby with teeth only just erupted and quite free from caries. One cannot exclude some local injury of the oral mucosa, but in those cases I have seen the usual history is of swelling with subsequent sinus formation in the mouth which does not suggest an osteitis resulting from a surface infection of the gum.

The last case I saw was that of a boy aged 2 years; there was a sinus in the palate midway between the incisor and the junction of hard and soft palate and another sinus opening on the gum at the cervical margin of the left deciduous central and extending upwards for about half an inch.

The incisor was quite free from caries, so were the other temporary teeth. I removed the incisor and scraped both sinuses. There was a temporary improvement but the sinuses still persisted. Under a general anæsthetic I then opened up the sinus on the gum and removed a piece of loose bone. The forming permanent central incisor was laid bare and I removed this tooth. The condition cleared up after this and has not recurred.

I am not sure that I did wisely in removing the unerupted tooth even although it was exposed and its crypt apparently directly connected with the sinus. Experience shows that developing teeth have extraordinary vitality and can go on living and developing under very adverse conditions. It might have been better to have been content with laying open the sinus and leaving the tooth. If it died its removal later would have been easy and at the most would have delayed slightly the progress of healing. The point I would emphasize in the treatment is that where the infection involves the body of the maxilla intervention should be limited and cautious. An unerupted tooth should not be sacrificed just because it happens to become exposed in laying bare a sinus. It should be left in order that one may see whether it may not survive. If the suppuration still persists any unerupted tooth which appears to be involved in the process can then be removed in order to provide drainage. But since there does not seem to be any risk of an acute development it is worth while to attempt to conserve the permanent teeth even though the final healing is delayed. The end-results seem to be good, provided that patience is shown and that a conservative surgical attitude is adopted. In those cases I have seen, there has been little permanent deformity of the maxilla, though usually in the course of the infection one or two of the developing teeth are lost by exfoliation, or removed in the course of treatment. My experience has been of those cases in which the osteomyelitis has affected the tooth-bearing part of the maxilla. Possibly other cases may take a different course and involve the orbit or nose and need more drastic treatment; and my generalizations based on those cases I have seen may not be justified. I should like to hear the experience of others on three points:—

(*a*) The pathology of the condition.
(*b*) The various forms which the condition may take and their surgical significance, and
(*c*) The treatment of the condition.

Tuberculous Infection of the Jaws.

My experience of these cases has been slight, but I have seen a few, through the kindness of my surgical colleagues at Great Ormond Street Hospital for Children. In all the cases I have seen the disease has occurred in the mandible. Is it known whether tuberculosis of the maxilla occurs? Other points on which I should like information are the following: Is this tuberculous infection connected with pre-existing dental sepsis? Is it grafted on an existing sepsis due to dental infection or may it occur in a clean mouth? Is it always secondary to tuberculous infections elsewhere or may it originate in the mouth as the primary infection? Experimentally it has been shown that it is possible for the tubercle bacillus to obtain entry into the bone through the pulp and the periodontal membrane, and I believe there is some clinical evidence that occasionally this path of infection may occur, though I cannot find the reference to it. Have any Members seen cases which might support this view? Seeing how common dental sepsis is in children and that the commonest source of surgical tuberculosis is from infected milk, it is theoretically possible that infection through the teeth or gums into the bone might occur. On this subject the Members of the Section of Odontology are seekers after information from their surgical colleagues.

Localized Suppuration in Connexion with the Jaws without Signs of Dental Infection.

From time to time I see cases in which localized suppuration in connexion with the jaws occurs which does not seem to be of dental origin. It is probable that many of these come under the care of the surgeon and are never seen by the dentist: an example of that defective liaison which handicaps both dental and general pathology. I should be glad to know whether others have seen such cases and can throw any light on their pathology. The commonest cause of an alveolar abscess is dental infection, chiefly through an infected tooth-pulp, but also through infection of the gum margin, in which case the pulp may be vital and the tooth even free from caries, though this latter variety of paradental suppuration is rare in children. A very careful examination is necessary before it is possible to say that a localized abscess perforating the alveolar bone is not due to a tooth. The fact that the teeth are free from caries does not exclude one of them being the cause, and a radiograph should always be taken. Even this may not be conclusive, for it is possible for a pulp to be dead and infected and the radiograph to show no signs. I make these points, for I have seen several cases of suppuration leading to a sinus in the mental region without any obvious signs of dental infection. In all these cases the teeth were free from caries. There was no sign of pyorrhœa and the gum margin was intact, ruling out the possibility of a paradental abscess.

An additional feature which the cases had in common was that the history was of long duration and that they had been under medical attention without the cause of the condition being discovered. In two of them a careful examination suggested that the pulp of one of the incisors was dead. In all of them a radiograph showed the presence of definite apical rarefaction round one of the lower incisors. The extraction of the affected tooth in all cases cleared up the sinus in a week or two. Here were a series of cases in which the dental cause was so obscure that it had been overlooked by the doctors in charge of the cases and it was not until they came under dental observation that the true nature of the condition was discovered. I have described this condition, though it does not come under the subject of this discussion, because it emphasizes the fact that a dental infection may be very obscure and that a careful examination is necessary before one can be certain that suppuration in connexion with the jaws is not due to an occult dental infection, even although a casual examination may seem to rule this out of court.

192 Pitts: *Oral Manifestations of Systemic Disease in Children*

Osteomyelitis of the Jaws following Scarlet Fever.

Recently Mr. Layton made a communication to the Section of Odontology[1] on a case of osteomyelitis of the maxilla following scarlet fever in a child. There is an excellent account of the condition, by Harold Austen, in Colyer's "Dental Surgery." The condition appears always to have been a rare sequel to scarlet fever, but even more uncommon now than a generation ago. The points I would like to raise are these: Is there any reason to suppose that this scarlatinal necrosis is a specific feature of the disease and due to the same streptococcal infection causing the scarlet fever? Or, as I would prefer to think, is the occurrence of necrosis in a sense accidental and its explanation is that a lack of oral hygiene, perhaps combined with a pre-existing dental sepsis and the lack of the normal cleansing of the mouth due to a slop diet, has so lowered the resistance of the oral tissues that necrosis readily supervenes? If this view be correct then the comparative rarity of necrosis following scarlet fever is explicable by reason of the greater attention paid to oral hygiene as part of the regular routine of nursing. In this sense the condition might be defined as my old teacher, Sir James Fowler, used to define the typhoid state, as that state into which no patient should ever be allowed to get.

Oral Manifestations of Congenital Syphilis.

The oral manifestations of congenital syphilis demand consideration. The ætiology of the Hutchinsonian tooth is still obscure. The fact that the dental stigmata are nearly always symmetrical, like the hypoplasia following such a disease as rickets, suggests that there must be some central factor affecting calcification and

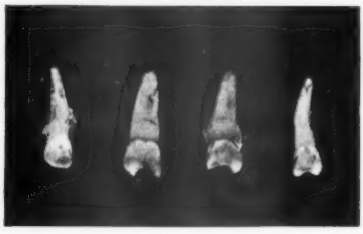

FIG. 1.—A photograph, natural size, of four upper deciduous incisors, showing notching due to caries.

producing the condition as opposed to a local factor. Yet very rarely does one see a case in which one central incisor shows the typical malformation while the corresponding tooth on the other side is normally formed. I am unable to offer any explanation of this curious fact. The statement has been made that occasionally the deciduous incisors may be affected by congenital syphilis and show the typical malformation. Personally, I am very sceptical about this. During the twenty years I have been connected with Great Ormond Street Hospital I have been closely on the look-out for a case, and though I have had the opportunity of seeing a large number of children with congenital syphilis I have never seen a case of Hutchinsonian incisors in the deciduous dentition. This is not to say that they do not occur, but it at least warrants a doubt. A hypoplasia of the milk teeth, not different in kind from that found in the permanent teeth in children who have had rickets and other diseases, may be found in syphilitic children, but this is a different matter, and except that

[1] See *Proceedings*, 1927, xx (Sect. Odont.), p. 48.

hypoplasia of the deciduous teeth implies a pre-natal condition and that congenital syphilis is one of the most important diseases affecting the fœtus, such a hypoplasia does not possess any special diagnostic significance. I would offer this explanation of the statement that Hutchinsonian deciduous incisors have been observed. In children of two or three years I have often noticed that when the upper incisors become carious, as they frequently do, the caries may start on the cutting edge and produce a notch. Superficially, such teeth might be taken to be Hutchinsonian teeth, but they are never peg-shaped and are quite different from true Hutchinsonian incisors (fig. 1).

In spite of the fact that the teeth in congenital syphilis still possess considerable diagnostic value I believe there is a good deal of confusion about them.

Many malformations which are not syphilitic are sometimes mistaken for them, and, conversely, I believe that the less typical forms of Hutchinsonian incisors are often overlooked. There are two features in a typical Hutchinsonian tooth (fig. 2):—

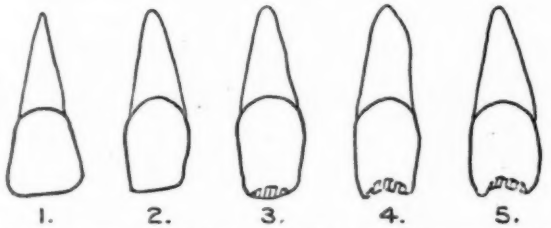

FIG. 2.—(1) Normal upper central incisor, showing typical divergence of sides from gum to cutting edge. (2) Minimal type of Hutchinsonian incisor; sides converge toward cutting edge which is straight. (3) Sides converge; small crescentic area of defectively formed enamel (potential notch), but no actual notch. (4) Sides converge; small actual notch present, and above it a larger potential notch indicated by stippled area. (5) Sides converge; actual notch increased in size at expense of potential notch. (These drawings are diagrammatic.)

(1) *An alteration in the architecture of the central incisor* so that the crown converges from the gum toward the cutting edge. Normally, the crown diverges from gum margin to cutting edge, so that the actual volume of a syphilitic incisor is considerably diminished.

(2) *At the cutting edge itself there is a crescentic area of badly formed enamel*, or the enamel itself may be completely absent, giving rise to a notch.

These two features are quite different from each other, and I would suggest that they are probably due to different causes. The first affects the architecture of the tooth, and must exist from the commencement of calcification up to the time when the crown is completely formed. The second feature exists to a variable extent and would appear to be a limited hypoplasia comparable to that occurring in rickets, as pointed out by W. H. Dolamore. My observations lead me to think that syphilitic incisors can be graded into four groups.

Type I.—A tooth showing the characteristic convergence of the sides towards the cutting edge. The enamel, as in all these teeth, is well-formed. The second feature, of crescentic hypoplasia at the cutting edge in this group, is absent. Such a tooth does not possess a high diagnostic significance, yet I would suggest that an alteration of an incisor from the normal splayed-out shape to the convergent type is one that, in my experience, does not occur except in congenital syphilis, and that its existence, even without other stigmata, should arouse suspicion enough to warrant further investigation. In the case of one child I saw only one permanent central was present. The boy attended my department for toothache. I noticed that this central showed a marked convergence on the sides toward the cutting edge, though this latter was perfectly straight. I sent him to be examined for the Wassermann reaction, and Dr. Nabarro reported that it was strongly positive. I have seen other similar cases and I believe that this type of tooth represents the minimal degree of the Hutchinsonian tooth.

Type 2.—In addition to the convergence of the sides there is a crescentic area of badly formed enamel but with no actual deficiency of tissue, so that there is no notch present. At a later stage after eruption this poorly formed tissue may break away and form a notch. This area of hypoplastic enamel might be termed a potential notch. The diagnostic value of this malformation is greater, for it approximates more closely to the description as given by Hutchinson and generally followed since.

Type 3.—In addition to the crescentic area of hypoplastic enamel or potential notch, there is a deficiency of enamel forming an actual notch. In other words the localized hypoplasia has been greater and there has been a complete absence of calcification of the central part of the cutting edge, leading to a definite notch, though this is smaller than the potential notch. The diagnostic value of this type is considerable and may become greater if the defectively formed enamel breaks away.

Type 4.—There is a greater extension of the actual notch at the expense of the potential notch, which, in conjunction with the convergent sides, gives the characteristic picture as described by Hutchinson. This type has the greatest diagnostic value. Types 1 and 2, though ill-defined, should still be looked upon with suspicion, for even trifles may be of value in the diagnosis of such a disease as congenital syphilis.

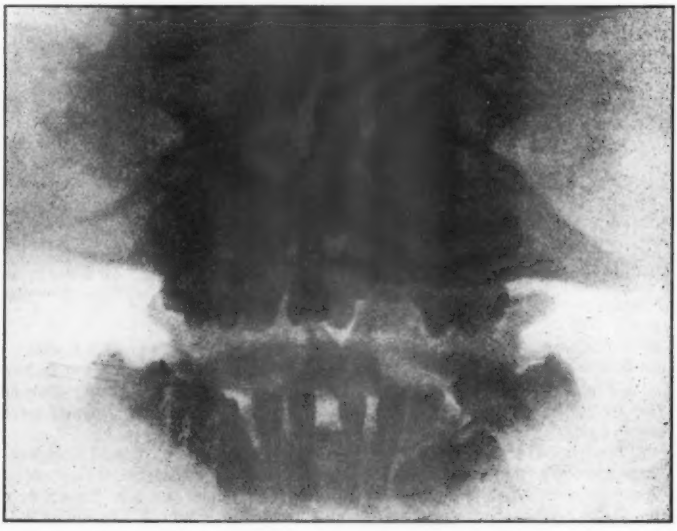

FIG. 3.—Radiograph showing an unerupted upper central incisor with well-marked notch. The incisor on opposite side has erupted and shows the same condition.

The statement has been made that the notch is produced after eruption by wearing away of the central part of the cutting edge. This is possible, and probably does occur in some cases, but I do not believe that it is the normal sequence of events. I have seen the notch present in well-defined form in teeth which have just erupted before any attrition could have occurred. I have also been able to demonstrate it in an unerupted central incisor (fig. 3).

This case was that of a child in Great Ormond Street Hospital for Children, with almost every sign of congenital syphilis. One central incisor was present with a well-marked notch. A radiograph showed that the central incisor on the other side, which had not erupted, possessed an equally well-marked notch.

The dome-shaped first permanent molars described by Moon have a high diagnostic value but not so great as the maxillary centrals. The molars are affected by so many other conditions and the shape may be simulated closely by hypoplasia due to other diseases. The association of Hutchinsonian incisors with Moon's molars forms a sign which is pathognomonic of congenital syphilis, and, in my view, is not produced by any other condition; but the occurrence of Moon's molars alone with normal centrals, though highly suspicious, needs confirmation by other signs, and in their absence, if the Wassermann was negative, I should hesitate to conclude that they were caused by syphilis, though I should certainly suspect it.

The statement has been made in some text-books that Hutchinsonian incisors are not specific to congenital syphilis and may be produced by other conditions. One might hesitate to say of any physical sign that it was absolutely specific of a particular disease, but I should certainly have said that a well-marked Hutchinsonian incisor was as near specificity as a sign of congenital syphilis as any physical sign can be. I have never seen Hutchinsonian incisors in their most typical form in which the diagnosis of congenital syphilis was not evident on other grounds.

One further point: Hutchinson made the statement that when perforation of the palate occurs the incisors are not affected. One does not now see many cases of perforation of the palate, but in those I have seen I have noticed that Hutchinsonian incisors were absent. It would be interesting to know whether others have observed this. If this generalization is correct what is its significance? Could it be taken as additional support for the hypothesis that there are two types of spirochætes with affinities for different tissues? I ask the question without venturing to propound any answer.

There are many other oral manifestations I should like to discuss but time does not permit. I will however offer a few brief comments in the hope of eliciting discussion and information.

Factors affecting Development and Eruption of Teeth.

The statement is made that rickets retards eruption. I presume this refers to the deciduous teeth about the eruption of which dentists know little, for we do not see children at an early enough age. I should like to hear the views of pædiatricians on this. What amount of delay may be taken to imply some pathological factor? My investigations of the dates of eruption of the permanent teeth showed that there was a wide variation within the range of normal eruption. Does the statement also apply to the permanent teeth? I have seen one or two cases of premature eruption of the permanent teeth in rickety children. What is the condition of the teeth in cases of generally retarded development? Is there any evidence to show that eruption is retarded in these cases. Again, what is the dental condition in cases of physical precocity? I saw one such case of a child of about four years with well-marked physical development under the care of Dr. Hutchison, in which the dental condition was at least three or four years in advance of her age. Have any similar observations been made? I would suggest that the dental condition should not be overlooked in these cases and that it should receive mention when these cases are described. If there was a relation between the dental condition and the physical condition, especially in those cases in which the latter depends on some endocrine abnormality, it would suggest that eruption of the teeth may also be regulated by the endocrine organs.

Cases of widespread deficiency of teeth are familiar to most dentists. They are of great interest, for there is usually an associated defect of other epithelial structures. The skin may be harsh and dry; the hair short and scanty; while cases have been recorded in which the sweat glands were absent. Such conditions remind one of the

196 Hutchison: *Oral Manifestations of Systemic Disease in Children*

examples of correlation of variation seen in some animals. Can any explanation, biological or pathological, be offered for this condition?

Lupus of the gums may occur, though it is rare. To the dentist unfamiliar with the condition it may pass unrecognized.

One case only has come under my care. I must admit to a failure to diagnose it, though to the dermatologist to whom I referred it the diagnosis offered no difficulties. In this case the ulceration extended between the upper incisors and eventually I removed these teeth and excised as much of the ulcerated tissue as I could. The wound healed up quite cleanly and the removal of the teeth allowed a much more effective treatment of the tuberculous area.

Pigmentation of the teeth, though rare, may occur in two diseases. The occurrence of green teeth as a sequel to infantile jaundice has been described by Dr. Thursfield. That of pink teeth in congenital hæmatoporphyrinuria is familiar, thanks to the careful studies of Garrod and Mackay.

Lastly, I would raise the question of vitamin deficiency and its relation to oral manifestations. Infantile scurvy is the classical example. One American writer has attributed follicular stomatitis in children to an absence of water-soluble B vitamin, and has recorded successful results upon giving tomato juice. During the Great War ulcerative stomatitis became very common in children in Middle Europe and assumed a quasi-epidemic form. Continental writers have suggested that a vitamin deficiency may have been a factor here. Is there any evidence to support this view?

I fear that my contribution consists chiefly of interrogations rather than information, but for that reason it may the better provoke a free discussion, of benefit to Members of both Sections.

Dr. Robert Hutchison.

It is a little difficult to know how best to open this discussion from the medical side. Perhaps the ideal plan would have been to describe the oral manifestations of general disease according to their ætiology, dividing them into the developmental, constitutional or metabolic, toxic, and infective manifestations, and so forth. This method of approaching the subject is possibly too pathological, and for practical clinical purposes it may be better to consider the different manifestations according to the structures affected. I shall deal, therefore, with general diseases as they affect the jaws, teeth, gums, tongue and lining membrane of the mouth respectively.

(1) *The Jaws.*—Affections of the jaw in general disease are more surgical than medical. General hypoplasia of the skeleton, however, such as occurs in some forms of infantilism, affects the jaws as well as the bones. It is tempting to speculate in this connexion as to the part played by endocrine disturbances. Overaction of the anterior lobe of the pituitary, for instance, causes an overgrowth of the lower jaw in acromegaly, and it is possible that pituitary deficiency may result in under-development of the jaw during the period of growth. In cases of dyspituitarism, however, such an under-development does not appear to occur. There is even less reason to believe that a deficiency of vitamin B in the diet can interfere with the development of the jaw, as some American writers have suggested. Rickets, on the other hand, may act in this way, although I think it does so to a less degree than might be supposed.

(2) *The Teeth* seem to be more affected by general diseases than the jaw itself. Their appearance in cases of rickets is delayed. In this connexion I should like to raise the question whether the teeth are not a better index of the real age of a patient than the ossification of the epiphyses. In a case of precocious puberty, for example, I have found the milk teeth still present at the age of three although the skeletal development was that of a child of twelve; in renal and other forms of infantilism,

I believe that the dentition is normal for the child's age, though the patient is otherwise much delayed in development.

Very important is the influence of general disease on the calcification of the teeth. This is well seen in rickets, as Dick has pointed out, and the experimental observations of Mrs. Mellanby show that it is due to a deficiency of vitamin A. The effect of inherited syphilis on the teeth is also well recognized. Anything, too, which impairs the general health may interfere with the laying down of enamel, and a groove more or less broad, or a succession of grooves with healthy enamel between, may mark attacks of grave illness during the time when that particular level of enamel was forming under the gum. Measles and pertussis seem specially apt to act in this way.

It is worth considering also whether variations in the reaction of the saliva in consequence of affections of the general health may not affect the enamel. Acidity of the saliva might be expected to do this, but to such variations in reaction little attention has been directed, so far as I know. Indeed, the whole question of the relation between departures from good general health and a predisposition to dental caries is very obscure. I cannot say that I have observed that there is any close relation between them. Apart from alterations in its reaction, an increased viscidity of the saliva, the result of a disproportionate "sympathetic" secretion might be expected to favour caries by interfering with the washing away of food particles. There is little doubt that such an increased viscidity is apt to occur in the case of nervous children.

Grinding of the teeth, which may proceed to such an extent as to cause them to be worn down, is a condition very familiar to all physicians, though its cause is often obscure. It seems sometimes to be merely a bad habit, but is also met with in many mentally defective children and in those who suffer from dyspepsia and perhaps from worms. It is usually practised during sleep, and was believed by the late Dr. Leonard Guthrie to be merely a sign of distressing dreams.

Staining of the teeth is a rare and curious phenomenon met with when there is the circulation of a pigment in the blood whilst the teeth are developing. "Pink" teeth are met with in this way in cases of congenital hæmatoporphyrinuria and "green" teeth in jaundice. Of congenital absence of teeth I have had no experience.

(3) *The Gums*, being highly vascular structures, easily become affected in diseases of the "hæmorrhagic" class, such as scurvy, the lymphatic leukæmias and purpura. In scurvy there may certainly be slight swelling and injection of the gums before the teeth actually erupt, but never to any great extent. The curious and rare condition of hypertrophy of the gums seems to be a purely local matter. I have only seen one case of it. It is possible, and even probable, that impairment of the general health may, by lowering resistance, favour the occurrence of gingivitis and pyorrhœa, but on this point, as on the influence of the general health on the production of caries, we have no very definite information.

(4) Hypertrophy of the *tongue* is met with in cretinism and to some extent in mongolism, and in the latter condition fissuring of the organ becomes pronounced as the child grows. The relation of furring of the tongue to conditions of general health is a large and obscure subject, but one not of any special interest to our dental colleagues. I would only say that the curious condition called "geographical" tongue which they may have noticed is not believed to have any significance.

(5) Affections of the *mucous membrane of the mouth* are, in my view, for the most part purely local and not indicative of any general disorder. This is certainly true of the common forms of stomatitis (catarrhal, maculo-fibrinous and thrush), although in the ulcerative and especially in the gangrenous form (noma) enfeeblement of the general health plays a large part, though I think it very doubtful whether vitamin deficiency is a factor. Mercurial stomatitis is very rare in childhood.

198 Parfitt—Cameron: *Oral Manifestations of Systemic Disease*

The eruption of some of the specific fevers such as chicken-pox is sometimes met with in the mouth, and Koplik's spots are, of course, a common phenomenon in measles.

Perforation of the *soft palate* may occur in the delayed form of inherited syphilis. This is often met with in association with interstitial keratitis. Whether the teeth are always affected as well I am unable to say.

Speaking generally, I would say that in my opinion the oral manifestations of general disease in childhood are, except as affecting the teeth, neither numerous nor important, just as I believe that the alleged effects of oral disease on the general health tend to be exaggerated.

Mr. J. B. Parfitt (Chairman)

said that there was a service the ordinary dental practitioner could render, namely, that of pointing out certain early manifestations of disorders of metabolism and development. The dental surgeon was treating large numbers of patients with caries, periodontal disease and dento-facial irregularities.

Caries appeared to be caused by certain micro-organisms, but while these might be necessary, they were not the all-sufficient cause, as seemed evident from a small case that came under his observation.

One of his own family was practically free from caries up till the age of 4, when she had an attack of influenza, with some inflammation of the oral mucous membrane, lasting about a fortnight. For about three months after this attack the child kept developing carious cavities in her teeth. These were filled, and after that time she once more became immune for a while.

Periodontal disease, although so common in adults, and, like caries, partly dependent on some constitutional condition, was rare in children.

Dento-facial irregularity due to errors of growth, was said by Mr. Chapman to be discoverable very early in life, even before the second or third year.

He thought that a dentist could often do great service by reporting to the patient's medical man any change in the immunity of a child to caries, or any indication—the earlier the better—that facial growth was not taking place along normal lines.

Dr. H. C. Cameron

said he thought that, so far as it was possible to put children into types—and types were mainly determined by inherited peculiarities, developed, perhaps, under the stress of faults in hygiene and of diet—one could make a distinction between two common types of children. In one type the teeth were almost habitually faulty, while in the other type, as a rule, the teeth were comparatively well formed, well erupted, and with good enamel. The type in which caries was liable to occur was, he thought, that type of child with a high content of lymph in the body, a round, plump child, often with high-coloured cheeks, with a considerable tendency to overgrowth of lymphatic structures—adenoids, tonsils, lymphatic glands—with a large appetite, a clean tongue and loose bowels. That was the type which especially showed a great tendency to catarrhal infections. He thought that the repeated catarrhal processes exercised intermittently a prejudicial influence on the complex activity of the enamel organ. In those children one often saw that circular caries of which Dr. Hutchison spoke, just as the nails were apt to show a white line indicative of some recent infective disturbance. It was common, too, to find the molar teeth erupted not vertically, but almost lying on their side, as if in obedience to some powerful inwardly-directed pressure, the result being that the opposed teeth met at their edges instead of surface to surface.

The second type of child, who seemed as a general rule to have good teeth, in many respects afforded a contrast. It was a child of intellectual inheritance, with all the metabolic instability associated with a family history of gout, migraine, asthma, eczema, etc.; often a pale, thin child, wasting its little body by the excess of its eagerness, frequently subject to bilious attacks, to cyclical vomiting, and to a host of nervous manifestations which had always interested him (the speaker). Their disorders were rather of metabolism than due to infection. Usually these children had good teeth, with good enamel.

The other question to which he would refer was that of the use of the jaws and the influence of that use on the satisfactory eruption of teeth. There had been much propaganda on this subject in infant welfare work, and many instructions were issued to mothers advising them to allow their children to bite on hard food. He thought the fault was seldom due to maternal ignorance. The child who developed well, who had good neuro-muscular control, who could walk early, and could use its hands early, would also use its jaws early and efficiently. The child whose infancy had been punctuated by catarrhal infections, who was slow to develop, who walked and talked late, would use its jaws late as well. It might be that the unsatisfactory dentition was partly due to the slowness with which the child learned to use its jaws. Corroborating that, perhaps, was the observation that children who had gross organic disease of the brain, who were diplegic, for example, who suffered from cerebral hæmorrhage at birth, or from severe hydrocephalus, nearly always cut teeth which decayed almost at once. Often in these cases the infantile suction was not replaced by the power to chew until the third, perhaps the fourth, year, and there was great difficulty in getting the children to take to solid food, because the incoördinate attempt to swallow a bolus was apt to bring on an attack of choking. While it was proper to give instructions to mothers to encourage their children to bite solid food, it was well to remember that the power to chew properly and to deal with such solid food was controlled by the nervous system, and was, in a sense, a gauge of the development of the nervous system in any particular child. Mere propaganda for early and efficient chewing could achieve little more than would propaganda for the promotion of early and articulate speech.

Dr. D. NABARRO

said that he would confine his remarks to the question of the teeth and palate in congenital syphilis. He agreed with Mr. Pitts' statement that different types or stages of teeth were to be seen in congenital syphilis. Those who talked of Hutchinsonian teeth had in mind the typical teeth which passed under that name, and which at once arrested one by their gross and obvious defects. Such teeth were uncommon, but there were teeth with much less marked characters of the Hutchinson kind which were not at all uncommon, and these, in his experience, were often overlooked. Therefore he did not agree with Dr. Hutchison when he said that the Hutchinsonian teeth did not matter very much, because one always had the Wassermann test to fall back upon. The latter statement was true, if the test were carried out, but it was not performed unless there was a suspicion that the child was the subject of inherited syphilis.

Mr. Pitts did not refer to the condition of the lower incisors. In his (the speaker's) experience, when there were marked Hutchinsonian upper incisors, the lower incisors, too, were very characteristic; they were small, their anterior surfaces were very convex, and there were wide spaces between the teeth, making them look like miniature tombstones in their settings, and they stood out in the jaw. Although they were usually associated with typical, pegged, Hutchinsonian teeth, he had seen them present without these or Moon's molars. In Moon's molars one

200 Nabarro: *Oral Manifestations of Systemic Disease in Children*

found degrees of it, sometimes with the domes tumbled in. In some children he had seen only one molar, or one on each side, affected in this way. As Mr. Pitts said, that was difficult to understand, just as it was difficult to understand why the right upper central incisor should be pegged and not the left.

Although statistics were often considered to be misleading—and it had been said that figures could be made to prove anything—he had nevertheless looked up the records relating to 208 cases of congenital syphilis affecting children of seven years of age and older. Records relating to children of lesser age did not give much help in regard to Hutchinsonian teeth, for he had never seen a typical Hutchinsonian tooth in the first dentition.

In the 208 children over seven years of age, 32 (15 per cent.) had typical Hutchinsonian teeth, either alone or in association with varying degrees of Moon teeth. This meant that 85 per cent. of the children had not typical well-marked Hutchinsonian teeth; 93 (45 per cent.) had normal teeth. The remaining 83 (40 per cent.) showed slight changes of the Hutchinsonian or Moon type. It was important to bear this in mind, as he had often seen a child in the ward or out-patient clinic in whom the peculiarity of the teeth had been overlooked. Dr. Jewesbury, at St. Thomas's Hospital, had recently analysed 200 cases which came to the Children's Department there, and had found that Hutchinsonian teeth were present in 14·5 per cent., and that in a further 9·5 per cent. of the cases the teeth were described as "irregular." But as he (Dr. Jewesbury) did not say how many of the 200 were over seven years of age, the two sets of figures were not properly comparable. Hutchinsonian teeth were not seen so frequently as they were sometimes thought to be.

With regard to ulceration of the palate, he had had twelve cases of this kind during the last twelve years; therefore it was not a common condition, though when seen it was quite characteristic. This ulceration of the palate, with, possibly, gummatous disease of bone beneath, was evidence of syphilis. The subjects of this palate disease ranged from 5½ to 12 years, but chiefly between the ages of 8 and 10. Only in one of them were the teeth of the typical Hutchinsonian character, and that particular child had bosses on the head, was deaf, and was suffering from interstitial keratitis and choroiditis; yet he looked healthy, and it was difficult to convince the parents that there was anything seriously the matter with him, and they required much persuasion to bring him up for treatment. In two others of the twelve cases there was wide spacing in connexion with the lower incisors. Three of the children had slightly suspicious molars. Hence he did not quite agree with Sir Jonathan Hutchinson's statement that the teeth were not affected when there was ulceration of the palate. Dr. Hutchison said these cases were usually associated with interstitial keratitis, but only two of these twelve children suffered from the keratitis; both were deaf.

From the point of view of treatment, the Wassermann was very resistant in these cases of palate ulceration. He did not know what significance that had. The Wassermann eventually became negative in four of the twelve, in the others it was resistant, though tested over a period of five or six years, and most of those children had had from twenty to forty injections of novarsenobillon.

Another point of interest concerned the cerebro-spinal fluid. He had examined this in eleven out of the twelve cases of ulceration of the palate, and it was negative in all except one case in which there was optic atrophy and hydrocephalus, and in both the blood and cerebro-spinal fluid the Wassermann was strongly positive.

Mr. F. N. DOUBLEDAY

reported notes of the following case:—

Recently he had in his out-patient department a woman who said that from childhood onwards she had suffered from ulceration in her mouth, which had been attributed to congenital syphilis. She brought with her two papers showing that at different times her Wassermann reaction had been tested and had proved negative. When he went carefully into the case they were unable to find any dental evidence to account for the condition, which prevented her using her lower jaw to any extent, and wearing the denture she had had made. The dressers were reminded that the mucous membrane of the mouth was merely part of the skin invaginated to meet the upper end of the œsophagus, and it was found that she had skin lesions also. On her being referred to Dr. Barber, of the Dermatological Department, he sent her back with the diagnosis that the disease was lichen planus, with unusual lesions of that disease in the mouth. On the following week she was again at his (the speaker's) out-patient department, and presented him with a piece of paper on which were written the names and addresses of three medical men, with the request that he would write to each of them and tell them they had unjustly accused her of having congenital syphilis, and stating the true nature of the disease.

He thought that Dr. Hutchison was a little inclined to underrate the significance of manifestations of disease in the mouth. In the case he had just narrated, the mouth was certainly a clue to the correct diagnosis.

He would mention a surgical condition which he had not heard referred to, so far, in the discussion. Children were sometimes brought who originally had abscess of temporary teeth, which infected the crypts of the permanent teeth. It was not osteomyelitis of the jaw, such as occurred in adults, but an infection of crypts of permanent teeth beneath. He hoped that some of his hearers had been more successful in the treatment of such cases than he himself had been. On several occasions he had cleared out the crypts of the permanent teeth, or this had been done by surgical colleagues, and the patients were sent away under the impression that they were cured. But they came back with swollen faces from residual infection, which it was very difficult to get rid of.

Reference had been made to ulcerative stomatitis, and he (the speaker) suggested that sufficient care was not exercised in recognizing in children ulcerative stomatitis associated with infection due to the *Bacillus fusiformis* and *Spirochætæ dentium*. In the war there was a definite epidemic of that infection, and it could only be diagnosed by making direct smears. When that diagnosis had been made, the infection easily yielded to treatment by chromic acid. And there were a certain number of cases of gangrenous stomatitis in which the bacteriology was obscure, and of those some had been shown to be due to the same infection.

He was also interested in what Dr. Hutchison said relating to the ductless glands and their influence on some conditions occurring in the mouth. He supposed that many practitioners had tried, in recent years, to influence calcium metabolism by giving preparations of ductless glands by the mouth, and had been uncertain as to whether those preparations produced a definite result. He thought that the treatment of children threw some definite light on that problem.

He referred to the case of a young woman, aged 18, who looked like a child of 7 or 8 years; she had long hair down her back, her stature was that of a child of about 8 years, and she was in the habit of playing with childish things. Her incisors, canines, and first molars had erupted, the premolars were not erupted. When ½ gr. of dried thyroid extract was administered by the mouth she began to grow, and the teeth erupted well.

And he had seen cases in which the jaws grew following the administration of anterior pituitary lobe extract.

In his view, one of the most serious manifestations of general disease in the mouth of a child was dental caries, and it was often directly induced by the advice given by

202 *Oral Manifestations of Systemic Disease in Children*

medical practitioners to mothers. Often mothers came to him quoting some medical practitioner as her authority for giving her child sweets to eat; the practitioner having said it was an easy way of raising the carbohydrate content of the diet. In adult out-patient departments it was found that many of the patients' illnesses were due to gastro-intestinal stagnation, largely brought about by their having overloaded themselves with sugar in childhood. Anyone who troubled to take cultures from mouths would see how streptococci throve on glucose broth. If children and young adults were given an excess of sugar, it remained undigested in the intestine, and as it supplied a good pabulum for streptococci to flourish upon, he therefore believed it to be a cause of many of the later streptococcal infections in adult life.

Dr. J. Kingston Barton

said that in accordance with the remarks made by the previous speaker he supported the view that much harm was done by the want of care in dietary instructions given by many medical men when prescribing for sick children. Without doubt undue amount of sweets and all kinds of glutinous sweet confections consumed by children supplied a most favourable soil for the growth of all the many streptococci that inhabited the mouth and nasopharynx, and thus favoured the onset of that dire disease, acute rheumatism, which crippled so many children. It was now twenty-seven years since Dr. Poynton and Dr. Paine[1] showed that a streptococcus was probably the actual real agent in the disease, and although the actual bacillus or coccus was not yet positively discovered, all clinical evidence pointed to such an organism being the *fons et origo* of acute rheumatism. He (Dr. Kingston Barton) said that he began practice in 1880, and after hearing a most stimulating paper on the wide question of degenerative changes in the teeth of children, read by Professor Magitot, of Paris, at the International Medical Congress held in London, 1881, he had always taken the greatest trouble to see that every child, as soon as it had obtained its first set of teeth (that is, when two years old), should be most carefully watched for any disease in the teeth. Whatever might be the matter with any child between the ages of 2 and 12 years, he had always insisted upon the teeth being, if possible, put in order before other elaborate investigations were made. He would like to hear from any younger member of the dental profession who had devoted attention to the development of the jaws, the true explanation of the small retracted lower jaw in so many modern children, which was unsightly in itself and led to much trouble later in life when artificial dentures had to be fitted. Was not this trouble possibly connected with hand-feeding, either from faulty bottles or teats in addition to the faulty foods? He would urge upon the younger generation of practitioners, especially the women doctors who were devoting their attention to diseases of childhood, to pay the greatest attention to the condition of the teeth in their bearing on all questions of ill-health in children. He agreed with Dr. Hutchison that in older people there had been an exaggeration of the connexion between bad teeth and many well-known diseases. But in children he did not think there could be too much care taken in keeping the teeth conditions perfectly free from all sepsis.

Mr. P. G. Doyne

said there was one point of association between general and dental conditions worth mentioning, namely, that existing between lamellar cataract and defective enamel of teeth. He had been struck by what Dr. Nabarro had said relating to the statistics of congenital syphilis and interstitial keratitis. His (the speaker's) own impression was that it was comparatively infrequent to see the typical Hutchinsonian teeth associated with interstitial keratitis. That, however, might be due to his own fault in not having recognized the more abstruse dental forms.

[1] *Med. Chirg. Trans.*, 1902, lxxxv, 211-242.

Dr. A. Livingston

said that it was admitted that there were two types of gum-margin inflammation. One of them was purely local in causation, namely, that with carbohydrate stagnation and neglect of hygiene; with this the discussion was not concerned. But there was another type—a gum-margin redness, which he regarded as a definite part of a generally lowered resistance. The physician or surgeon who saw it would find it accompanied by constipation, or it might be diarrhœa or other systemic upset. Occasionally it was associated with one of those unknown pyrexias of children which caused so much trouble. In the kind of case to which he was referring there were four causes, one, or all of which might be in operation: (1) General lowered resistance; (2) calcium shortage; (3) vitamin and sunshine deficiency; and (4) a fusiform spirochæte infection. Sometimes, and especially at that time of the year, the condition progressed to the stage of true ulcerative gingivitis or stomatitis. All those causal conditions were very common at the end of the war, together with the lowered resistance and the acid and calcium shortage played the greater part in causation.

Mr. Doubleday had said that he had found himself only able to diagnose the condition of ulcerative gingivitis by means of direct smears, but he (Dr. Livingston) thought the smell should be enough.

Mr. Pitts had mentioned the group of cases in which there was no ascertained cause for a localized dental infection. Those cases were very rare; he could himself recall only three. All those patients gave a history of very slight trauma in the remote past, with the presence, at that time, of a lowered resistance. The three cases just referred to occurred after efforts on the part of the children to bite string.

The teeth and gums of children were of supreme importance. The teeth and jaws of a child aged 6 years gave that child's past history; the mucous membranes and the gums gave more the present history.

Dr. A. A. Moncrieff

said there were two points on which he would like information. Mr. Pitts and Dr. Nabarro laid stress upon the fact that the deciduous teeth never showed Hutchinsonian characters. In Paris, Professor Marfan said that rickets was caused in 90 per cent. of cases by congenital syphilis, and demonstrated cases of children under 6 years of age showing a notch in the central incisor teeth. Could rickets cause a notch in the central incisors? Or was there some other causative factor in addition?

And was it true that in congenital syphilis the teeth erupted at an earlier age than normally, namely, the first of them at about the third month? If so, what was the cause?

Mr. Pitts (in reply)

said he had been interested in many of Dr. Hutchison's remarks. The point about the defective use of the jaws and the bearing of that on their size was of great interest. Many cases suggested that well-formed jaws might exist, though poorly used. He had seen two cases of ankylosis of the jaw, both of which had existed from an early time. In the lower jaw in both cases there was, as would be expected, much under-development, but the upper jaw was well-formed; both the children were well nourished.

What Dr. Hutchison said about the acidity of the saliva was also very interesting. He had never investigated that in children, but he had always had the idea that it might be a factor of considerable importance in the causation of caries.

On the question of the grinding of the teeth by children, he had no contribution to offer; he would like to know more about it.

204 Pitt—Hutchison: *Oral Manifestations of Systemic Disease*

Dr. Hutchison mentioned the question of diffuse hypertrophy of the gums, which was occasionally met with in children, which he regarded as a purely local condition. Heath described that condition many years ago, and said that in some cases there was a hereditary history. Recently a writer in America described a series of cases which occurred in one family, thus showing the hereditary nature of the condition. Only two cases of the kind had occurred under his (the speaker's) care, and in neither was there any question of heredity.

He had seen many cases of hypertrophy of the tongue in mongols, but in most cases the jaws of mongols were well formed. Many writers had laid stress on the action of the tongue as a spreading factor in promoting the action of the jaws; but in two cases of mongols with large tongues there was extreme irregularity of the deciduous teeth.

He was sorry that no speaker had mentioned the question of the high palate, which was often stated by neurologists to be a stigma of mental degeneracy. He would have liked to have learnt whether physicians present considered that there was any truth in that view.

Dr. Cameron's description of two types of children was very interesting, and he (the speaker) would be on the look-out to see whether he could find evidence of a similar nature.

In answer to Dr. Moncrieff, he (Mr. Pitts) gathered that in France the view as to causal influence of syphilis in many conditions was carried to an extreme degree, which was not supported by observers in this country. He suggested that notched temporary teeth were not due to syphilis or rickets, but were, rather, examples of caries, like the specimens of which he showed illustrations.

Dr. Hutchison (in reply)

admitted that sugar might be expected to be a factor in the production of caries of the teeth, but sugar was an extremely valuable food for the child, and to get the benefit of it, it seemed almost worth while even to run the risk of some caries. To state that sugar caused gastro-intestinal stagnation was to state what he believed to be untrue, as he considered there was no such condition, and for sugar to be absorbed imperfectly and remain in the alimentary canal was unthinkable, as sugar was one of the most rapidly absorbed of foods.

It was understandable that the child should not acquire pyorrhœa as easily as the adult, since it was not until adult life that the gums receded sufficiently for pyorrhœa to attack them.

One speaker described a new variety of gingivitis—a marginal redness, which apparently owned a multiform pathology, but he (Dr. Hutchison) was always sceptical about such a plurality of causes.